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Growth Through Agricultural Progress

The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Crops Research Division serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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ACCEPTANCE OF MANUSCRIPTS

The increase in the volume of pertinent material offered for publication in the Plant Disease Reporter has made it necessary to limit the subject matter and the length of articles accepted. The subject matter should emphasize new things in plant pathology, such as new records of disease occurrence, serious outbreaks and epidemics, conditions affecting development of plant diseases, techniques of investigation including instrumentation, new discoveries in control including new materials and their evaluation. Manuscripts will be limited to 12-double-spaced typed pages, including tables, graphs, and photographs. Because of reproduction costs photographs should be kept to a minimum. Insofar as possible, material should be presented as graphs rather than tables. Papers cannot be accepted for publication that report routine control experiments, reviews, bibliographies without annotation, results of routine surveys, mere summaries or lists of plant diseases. By following this procedure we hope to continue publishing all articles promptly.

Paul R. Miller

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SO-CALLED BEET LATENT VIRUS IS A BACTERIUM[1]

C. E. Yarwood, E. C. Resconich, P. A. Ark, D. E. Schlegel, and K. M. Smith

Summary

Attempts to purify and characterize "beet latent virus" as a virus were unsuccessful. Bacteriological isolations from cowpea leaves infected with "beet latent virus" have consistently yielded a motile bacterium which is probably Pseudomonas aptata. When pure cultures of this organism were inoculated to cowpea, lesions and systemic infections resulted which were similar to those produced by "beet latent virus." Some similarities and differences between the pathology of this bacterium and of authentic viruses are described.

INTRODUCTION

"Beet latent virus" was described by Smith in 1950 (16, 17, 18). It was manifested by lesions which resulted when juice from normal-appearing leaves of red beet, sugar beet, and mangold stecklings in England was rubbed onto cowpea and other plants. Subsequently it was found in Holland and California (19).

The features of this disease which suggest that a virus is the cause, are: 1) the supposed occurrence of the pathogen in beets and mangolds without symptoms, 2) the production of virus-like lesions on cowpea and other plants inoculated with non-filtered juice, 3) the ease of transmission by methods routinely used for viruses, and 4) the production of systemic infections of cowpea (17). Smith (17, 19) and Yarwood (22) pointed out certain peculiarities of this pathogen, among which were: 1) the successful inoculation of roots without the aid of mechanical injury, 2) the prompt elimination of the pathogen from roots by mercuric chloride, 3) the ease of sedimentation of the pathogen in the centrifuge, 4) the inability to find virus-like particles in the electron microscope, and 5) the decay of fruits as a result of inoculating them with the pathogen. It was therefore desirable that the true nature of this pathogen be established.

The early work was performed under the tentative assumption that the so-called beet latent virus was actually a virus. Routine virological methods were used. While this assumption proved wrong, the information secured is considered valid and illustrates some of the similarities and differences between a bacterial and a virus infection. The abbreviation BLV for the pathogen will be maintained, along with TMV for tobacco mosaic virus and TNV for tobacco necrosis virus.

RESULTS

Symptoms: BLV was characterized as an infection which was common and latent in beets but pathogenic to cowpeas. Inoculation of cowpea primary leaves by abrasion results in virus-like local lesions (Figs. 1, 5, 6) and systemic infection (Figs. 2, 3, 4). Local lesions were about 0.5 mm in diameter with (Fig. 1) or without (Figs. 5, 6) chlorotic halos. Systemic infections were manifested as necrotic (Figs. 2, 4) or chlorotic (Fig. 3) local lesions and distortion of the secondary leaves and occasional collapse of the petioles. Systemic infections occurred on about three-fourths of the plants which were inoculated on the primary leaves, and occurred more frequently when inoculum was taken from systemically infected plants than when taken from plants with only local infection. Inoculation of the stems of cowpea plants commonly resulted in systemic infection followed by death.

Host Range: Smith (19) reports BLV in Beta vulgaris, Vigna sinensis, Tetragonia expansa, Brassica oleracea and Phaseolus vulgaris. Most of these have been confirmed in this study. Cucumis sativus, Lycopersicum esculentum, Helianthus annuus, and Nicotiana tabacum are added as hosts on the basis of lesion formation following inoculation of leaves, Beta vulgaris, Brassica rapa and Daucus carota on the basis of lesion formation on slices of roots, Solanum tuberosum on the basis of lesion formation on cut slices of tubers, and Cucumis sativus, Cucumis melo, Malus sylvestris, and Pyrus communis on the basis of lesion formation and decay on cut slices of fruit.

Attempted Isolation as a Virus: Numerous attempts were made to effect at least a partial purification of the beet latent virus. Both density gradient centrifugation techniques and standard low-high-low speed centrifugations were tried with little success. Various modifications

[1]Aided by a grant (G 9820) from the National Science Foundation.

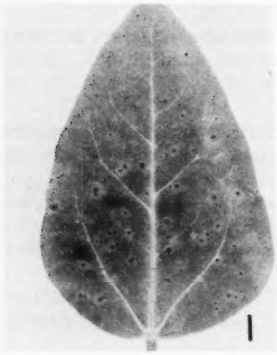
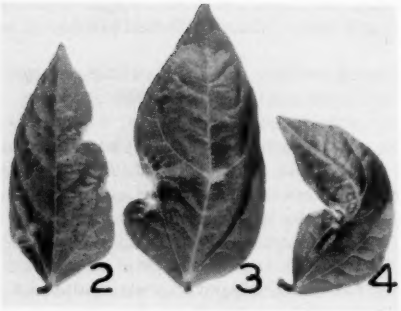


FIGURE 1. Lesions and chlorotic halos on primary cowpea leaf 4 days after spray inoculations with agar culture of BLV bacteria (Pseudomonas aptata).



FIGURES 2, 3, 4. Systemic symptoms consisting of necrotic and chlorotic lesions and distortion of secondary leaves of cowpea 18 days after the primary leaves of these plants were inoculated with BLV bacteria.

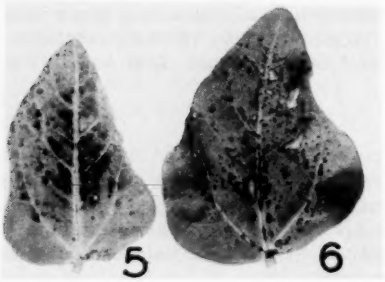


FIGURE 5. Lesions and injury by metabolites on primary cowpea leaf 5 days after abrasive inoculation with a water suspension of a pure culture of BLV bacteria on potato-dextrose agar.

FIGURE 6. Lesions on primary cowpea leaf 5 days after abrasive inoculation with a pure culture of BLV bacteria. This leaf was washed after inoculation and the leaf in Figure 5 was not.

Table 1. Relation of abrasion and moist chamber incubation to infection with BLV bacteria.

Method of inoculation	Number of lesions[a]					
	Plants incubated on dry bench			Plants incubated first 24 hours in a moist chamber		
	Trial 1	Trial 2	Trial 3	Trial 1	Trial 2	Trial 3
Spray	1	0	130	120	0	106
Ordinary abrasion	39	103	638	256	312	564
Abrasion followed by quick drying	56	42		242	26	
Abrasion followed by washing	30	30	1096	44	44	776

[a]Trials 1 and 2 were under ordinary greenhouse conditions. Trial 3 was under higher humidity in the greenhouse area but no free water was observed on the leaf surfaces.

of these techniques including the addition of ethylene-diamine-tetraacetic acid, detergent, proteins, and so forth during purification did not improve the results. In almost every case most of the infectivity, as determined by inoculation to cowpea leaves, was found in the first slow speed centrifuge pellet. Even when crude sap was layered directly on a density gradient system (with no prior slow speed centrifugation for clarification purposes) most of the infectivity went to the layer containing the cell debris and chloroplasts. Attempts to pass the virus through a bacteriological filter were unsuccessful, as would be expected on the basis of the behavior of BLV in the centrifuge.

Electron Microscopy: Although purification of the beet latent virus was not effected, the possibility existed that electron micrographs might show some particles which could be associated with the disease. Crude juice from infected cowpea leaves and beet roots was diluted to 1/100, 1/1000, 1/10,000 and examined with the electron microscope. No virus-like particles were found in any preparation. Occasional bacterial cells were seen but their significance was not appreciated.

Treatment with Phenol: Release of infectious nucleic acid following treatment of diseased tissues with cold phenol is a unique property of a limited number of viruses, not all of which can be purified readily (13). Accordingly, diseased tissues were homogenized in the cold in four times their weight of a 1:1 mixture of 1% tetra-sodium-pyrophosphate and water-saturated phenol. The aqueous and phenol phases were separated by centrifugation. Inoculations of cowpea leaves with dilutions of the aqueous phase were negative.

Isolation as a Bacterium: Pieces of diseased cowpea leaves were separately macerated, diluted, and streaked on potato-dextrose agar. The bacilli from the resultant colonies were actively motile in 24-hour cultures on broth, but no motility was detected in 48-hour slants of potato-dextrose agar. When two mass cultures on agar, six single colonies on agar, five transfers from single colonies on agar, and three broth cultures were separately used as inoculum on cowpea, lesions similar to those produced by inoculum maintained continuously in leaves resulted. The bacterium isolated from these leaves was similar to those of the original isolation.

Routine bacteriological study of the bacterial isolates from "beet latent virus" showed their close similarity to Pseudomonas aptata as originally described by Brown and Jamieson (6), and later by Ark and Leach (1) from sugar beets in California. The isolates of the present study were compared with an old isolate of P. aptata obtained from the American Type Culture Collection and representing a culture deposited by P. A. Ark in 1946. Parallel observations of morphological characteristics of all isolates revealed no significant differences. Pathogenicity trials on cowpea, sugar beet, and tomato plants showed that on first inoculation the 1946 culture of P. aptata caused fewer and smaller lesions on the inoculated leaves than did the recent isolate, but after one passage of the 1946 isolate through cowpea, the lesions caused by the two cultures were similar.

Pathology: Occurrence on beet. Since a bacterium is shown to be the cause of this disease, the features of the disease which might indicate a virus as the cause, previously listed, should be re-examined.

The apparent latency of this infection in beet and mangold does resemble latent infections with several authentic viruses on miscellaneous hosts (3). The general phenomenon of bacteria in or on healthy tissues is discussed by Hollis (11), and the specific example of pathogenic Pseudomonas mors-prunorum on healthy leaves of stone fruits has been reported by Crosse (8). We feel that the occurrence of pathogenic bacteria on or in normal-appearing beet leaves is not surprising.

Virus-like Lesions: While BLV produced virus-like lesions on cowpea and other hosts, virus lesions are not highly specific and lesions which could be confused with virus lesions are produced by certain bacteria (10, 20) and fungi. No comparative study of lesions produced by fungi, bacteria, and viruses seems to have been reported. In this study lesions of BLV on cowpea were much more variable in diameter than were lesions of tobacco necrosis virus on cowpea. The mean and standard deviation of 21 discrete BLV lesions at 7 days after abrasive inoculation was 0.425 ± 0.32 mm in their greatest diameter while for 21 TNV lesions also produced on cowpea by abrasive inoculation was 2.17 ± 0.58 mm. The coefficient of variability was thus 75% for BLV and 27% for TNV for comparable lesions on the same host. Some of this variability of BLV lesions was due to abrasive inoculation. In another comparison, BLV lesions produced by spray inoculation (Fig. 1) were 0.35 ± 0.13 mm whereas lesions produced by abrasive inoculation were 0.34 ± 0.33 mm (Fig. 6), or the variability due to abrasive inoculation was 2.5 times that from spray inoculation.

BLV lesions were also more sunken, less circular, more oily and shiny in appearance and

had more ragged edges than TNV lesions. BLV lesions were commonly surrounded by large but faint chlorotic halos (Fig. 1), whereas TNV or TMV lesions had no such halos.

Systemic Infection: The systemic infections which resulted in cowpeas from inoculation with BLV (Figs. 2, 3, 4) are not considered specific for viruses. Ground suspensions of lesions, collapsed petioles, and normal-appearing roots were shown to be infective, but the bacteria could not regularly be isolated from chlorotic lesions.

Abrasive and Moisture Effects: The transmission of an infection by methods used for virus transmission is no reliable indication that a virus is involved. The distinctive features of inoculations with viruses and bacteria are that for successful inoculation with a virus abrasion of the host is a necessary or standard procedure but a moist chamber incubation is not necessary or desirable (21), whereas for successful inoculation with a bacterium abrasion is usually not necessary but overnight or longer incubation in a moist chamber is. However, abrasion of leaves before or during inoculation may greatly increase infection with some fungi (4, 7, 15, 23) and bacteria (5, 12). In this study both abrasion of the leaves and incubation of plants in a moist chamber after inoculation aided, but was not essential, for infection (Table 1). Both rapid air drying and rinsing of leaves after abrasion decreased infection but inconsistently so. All the above evidence indicates that BLV is not a virus since abrasion of leaves is essential for most virus inoculations, since incubation of inoculated plants in a moist chamber reduces infection with viruses, and since quick drying and washing after inoculation increase virus infections (21).

Phosphate Effect: When 1% K_2HPO_4 was added to inoculum of BLV the number of lesions on cowpea was less than when no phosphate was used, whereas phosphate increased infection of cowpeas by all viruses studied (21). In three tests by twin leaf methods, the numbers of BLV lesions with and without phosphate were 109 and 299 respectively, whereas with TNV the comparable numbers were 380 and 33. In some trials inoculation of BLV with phosphate failed, whereas the controls gave good infection.

Mercury Effect: No recent work on the effect of mercury on BLV has been done, but the original finding (22) that the infection was completely eliminated when infected roots were dipped in 1-1000 $HgCl_2$ for 10 seconds was a major bit of evidence that BLV was not a virus.

Infection via Transpiration Stream: When cowpea plants growing in soil were cut at the hypocotyl and the cut hypocotyl dipped in inoculum, BLV lesions formed in a few days on the stems, veins of young primary leaves sometimes became necrotic, and distortion of the leaves sometimes resulted. This behavior is considered atypical of viruses and several trials of this method of inoculation with TMV and TNV have failed. However Schneider and Worley (14) have reported infection of beans with southern bean mosaic via the xylem.

Decay of Storage Tissues: BLV multiplied with symptoms on cut slices of beet root, turnip root, radish root, carrot root, potato tuber, pear fruit, apple fruit, cucumber fruit, and melon fruit. On pear, cucumber, and melon a soft rot usually resulted, and on beet a dark hard decay. On beet and cucumber slices, well defined lesions resulted at low concentrations of inoculum, and at 1-1,000,000 dilution of infected cowpea leaves, six lesions resulted on two cucumber slices, while at 1-100,000 dilution the lesions were too numerous to count. When dilutions of these decayed fruits or roots were used as inoculum, typical BLV lesions resulted on cowpea leaves.

This behavior of BLV on storage tissues is considered atypical of any virus infection, though the behavior of most viruses on these same substrata is not known. But TNV has been shown in this study to multiply abundantly but erratically on cucumber slices.

Injury by Metabolites: Another apparent difference between virus and bacterial lesions is that in greenhouse inoculations more injury due to metabolites of the pathogen results with bacterial infections than with virus infections. When cowpea leaves were inoculated by abrasion with BLV inoculum from cowpea leaves, beet roots, cucumber fruits, agar cultures and broth cultures, and more especially the latter four, general injury (Fig. 5) occurred on the cowpea leaves, and this injury was not specifically associated with lesions. No such injury to cowpeas resulted from TNV inoculum from cowpeas, or with most viruses from miscellaneous hosts. Also inocula consisting of the above substrata but without BLV, produced no such injury. This injury from BLV was eliminated or greatly reduced if the leaves were washed, or quickly dried after inoculation.

Another evidence of host injury by metabolites of the bacteria was the halos produced around some lesions (Fig. 1). This is characteristic of some bacterial lesions (9) but less so of virus lesions.

DISCUSSION

The confusion of a bacterium with a virus as the cause of plant disease has occurred before, but merits further discussion here. A virus is only reliably identified when a particle with one dimension smaller than 200 mμ is shown to be the cause of disease (2). But virus particles are frequently difficult to find and many viruses have been correctly identified, initially at least, without satisfying this criterion. Inability by ordinary microscopic and cultural methods to find a pathogen of a transmissible infection is usually reliable evidence that a virus is the cause. Graft transmission has been useful in identifying many viruses of fruit trees. Symptoms alone are useful in that a transmissible systemic mosaic is likely to be caused by a virus. The identification of a virus on the basis of the production of virus-like lesions following artificial inoculation is here considered tentative and not reliable, though the production of local lesions is considered a very useful method of assay of viruses which have been established as such by more stringent criteria.

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PEAR RUST (GYMNOSPORANGIUM FUSCUM) IN NORTH AMERICA[1]

Wolf G. Ziller[2]

Abstract

The establishment in North America of the trellis rust of pear, caused by Gymnosporangium fuscum DC. (G. sabinae Dicks. ex Wint.), is reported for the first time. G. fuscum has caused local damage to pear (Pyrus communis) trees in Victoria, British Columbia. Presumably the fungus was introduced on juniper seedlings from Europe 15 years ago. The taxonomy, life cycle, importance, and control of the rust fungus are outlined.

Trellis rust of pear, caused by the fungus Gymnosporangium fuscum DC. (G. sabinae Dicks. ex Wint.), has become established in a localized area in Victoria, British Columbia. Although the introduction of the disease is believed to have occurred approximately 15 years ago, not much attention was paid to it because it was mistaken for a common juniper rust caused by G. clavariaeforme (Jacq.) DC. In May 1960, however, telial states were discovered on ornamental juniper (Juniperus sabina vars.) less than 50 feet from the same pear trees that were heavily rusted in previous years. Examination confirmed the rust on both hosts to be Gymnosporangium fuscum, "the most common and serious pear disease in Europe due to a species of Gymnosporangium" (3). To the best of the author's knowledge this is the first record of G. fuscum in a locality outside of its native Europe, Asia Minor, and North Africa (10, 14).

In 1932, and again in 1933, a rust believed to be caused by G. fuscum was collected on imported ornamental junipers in a nursery at Winona, Ontario, but no rust on pears was noted in the vicinity of the junipers. "G. fuscum seems never to have become established at Winona" (7).

In October 1960 the rust was collected on Bartlett pear at Lafayette, California by Mr. Milton R. Bell, Farm Advisor; it was subsequently identified as G. fuscum by Professor George B. Cummins, Purdue University, and the identification was verified by Dr. Frank D. Kern. Further details of this latest discovery of the trellis rust will be published elsewhere.

Imported nursery stock of juniper infected with G. fuscum has been intercepted repeatedly at United States and Canadian ports of entry in past years (7).

TAXONOMY AND DISTRIBUTION

The trellis rust fungus has been known for centuries. Its distribution has been limited to Europe, Asia Minor, and North Africa. The knowledge of its parasitism, requiring both pear and juniper as alternate hosts, is as old as the knowledge of heteroecism in the rust fungi (95 years).

Pears are susceptible to at least 10 species of rust fungi all belonging to the genus Gymnosporangium (12). The following description, condensed from Gäumann (10) and Kern (11, 12), contains only characteristics that distinguish G. fuscum from the remaining nine species of Gymnosporangium recorded on pear.

GYMNOSPORANGIUM FUSCUM DC., Fl. Fr. 2, p. 217. 1805.

G. sabinae Dicks. ex Wint., in Rabh. Krypt. Fl. 2(1), p. 232. 1882.

Pycnia and aecia on yellow to bright-orange, tuberculate leaf spots (Figs. 1 and 2) swollen twigs (Fig. 3), or fruit of pear. Pycnia 170-190μ wide x 150-170μ high. Aecia acorn-shaped (balanoid), rupturing along the sides (thus the name "trellis rust"), the apex remaining conic, 0.5-1 mm in diameter and up to 6 mm high (Fig. 4); side-walls of peridial cells verrucose; aeciospores 20-28 x 25-35μ, light chestnut-brown. Telia conic or laterally compressed, dark brown, up to 5 mm wide and high (Fig. 6), on spindle-shaped enlargements of mostly the larger branches of juniper (Fig. 5).

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[1]Contribution No. 710 from the Forest Biology Division, Canada Department of Forestry, Ottawa. The cooperation of Dr. Frank D. Kern, Pennsylvania State University, University Park, Pennsylvania, and Mr. I. L. Conners, Research Branch, Department of Agriculture, Ottawa, Canada, is gratefully acknowledged.

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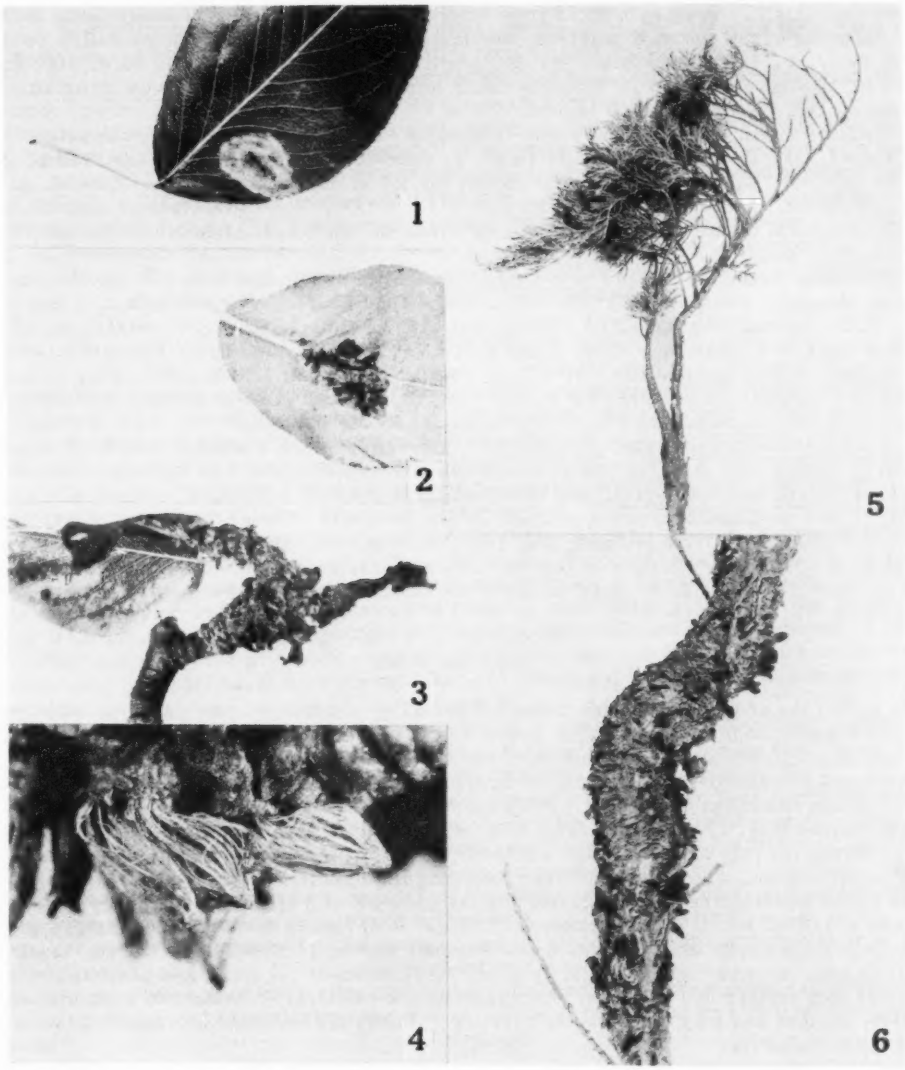

FIGURE 1. Pycnia on the upper leaf surface.
FIGURE 2. Aecia (roestelia) on the lower leaf surface below pycnia.
FIGURE 3. Roestelia on pear twig and petiole, from perennial infection.
FIGURE 4. Close-up of the balaniform (acorn-shaped) roestelia on a tuberculate area of a pear leaf. Note characteristic lateral dehiscence of the peridium.
FIGURE 5. Fusiform swelling of juniper branch caused by perennial mycelium of the pear rust fungus. The telial horns have sloughed off.
FIGURE 6. Same as Figure 5 but showing the telial horns before they expand and gelatinize.

Hosts for the aecial state - Pyronia danieli (Winkl.) Rehd. (=Cydonia oblonga Mill. X Pyrus communis L., graft hybrid), Pyrus [3]amygdaliformis Vill., P. [3]communis L. (cultivated varieties of the common pear), P. betulaefolia Bunge, P. elaeagrifolia Pall., P. orientalis Pall., P. [3]michauxii Bosc. (P. amygdaliformis Vill. X. P. nivalis Jacq.), P. [3]nivalis Jacq., P. lindleyi Rehd. (as P. sinensis Lindl.), P. syriaca Boiss., P. salicifolia Pall., P. [3]tomentosa DC., P. ussuriensis Maxim.

Hosts for the telial state - Juniperus [3]chinensis L., J. c. var. pfitzeriana Spaeth., J. communis L. (6), J. c. var. saxatilis Pall., J. excelsa Bieb., J. foetidissima Willd., J. oxycedrus L. (6), J. phoenicea L., J. [3]sabina L., J. s. var. tamariscifolia Ait., J. s. var. variegata (West.) Audib., J. [3]virginiana L.

Geographical distribution: Europe, Asia Minor (5, 6), North Africa (10), Canada (Victoria, B.C.), and U.S.A. (Lafayette, Calif.).

Specimens Examined -- On Pyrus communis: three specimens from Europe (Rathay 376, Austria; Mougeot, France ?, DAOM[4] 58712; and a specimen from Cyprus); Sept. 1945, Victoria, B.C., pycnia only (DAVFP[4] 12001); Sept. 1959, Victoria (DAVFP 11448); Dec. 1959, Victoria (DAVFP 11895); May 1960, Victoria (DAVFP 12468); Aug. 1960, Victoria (DAVFP 12469); Sept. 1960, Victoria (DAVFP 12319, Figs. 1, 2, and 4; 12320; 12321, Fig. 3; and 12322); Oct. 1960, Victoria (DAVFP 12357, used as inoculum in experiment); Oct. 1960, Lafayette, Calif., U.S.A. (DAVFP 12470, collected by M. R. Bell).

On Juniperus chinensis var. pfitzeriana: interception # 21, Toronto (DAOM 38689).

On J. sabina var. tamariscifolia: June 1960, Victoria (DAVFP 12000, Figs. 5 and 6).

On J. sabina var. variegata: May 1960, Victoria (DAVFP 12465).

On J. sabina: One European specimen, J. B. Mougeot, France ?, DAOM 58713; four specimens intercepted from Holland; May 1932, Winona, Ont. (DAOM 1721); May 1933, Winona, Ont. (DAOM 2019); identification of last two specimens is uncertain, for the rust was not found on pear (7), and with the destruction of the infected plants it seems not to have become established.

LIFE HISTORY

In spring the young pear leaves become infected by windborne basidiospores, which originate from the telial horns on infected junipers in the vicinity. These basidiospores are delicate and short-lived, and infection of the pear leaves can therefore take place only if the juniper hosts are nearby, within a radius of approximately 1000 feet (2). From 13 to 17 days after infection has taken place (10), pycnia appear on yellow blotches on the upper surface of the pear leaves (Fig. 1). Fruit, twigs (Fig. 3), and branches of pear may also become infected. Branches may be girdled and killed (4), or the fungus may overwinter in them as perennial mycelium. During the summer following the infection of pear, swellings begin to appear in the infected areas, particularly on the yellow leaf blotches directly below the pycnia. On these swellings aecial horns ("roestelia", Figs. 2-4) finally develop which mature and begin to disseminate their aeciospores in late summer and fall. These aeciospores, like the basidiospores, become airborne after they are disseminated; but unlike the basidiospores they can infect only juniper and not pear. Aeciospores are relatively resistant to temperature and moisture changes and may therefore be carried by wind currents for hundreds of miles without losing their viability.

In fall alternate hosts of the trellis rust, species of juniper, become infected by the windborne aeciospores. The disease remains hidden in juniper and cannot be identified for approximately 18 months after infection has occurred. It is during this initial period that trellis rust of juniper escapes detection. Finally in spring, 18 months after infection, the first crop of telial horns develops on the infected juniper branches (Fig. 6). The rust fungus is then established permanently in the juniper branches and will produce a new crop of telial horns from the same branch canker every spring, without apparent damage to its host. With the infection of pear by basidiospores, released from the first crop of telial horns on juniper, the 2-year life cycle of the trellis rust is completed.

DAMAGE AND CONTROL

Reference to damage by trellis rust is numerous in the literature; over 50 reports from

[3]Susceptibility proven by inoculation experiments (10).

[4]For abbreviations of names of herbaria Lanjouw and Stafleu (13) are followed.

19 countries have been reviewed. Eradication of all rust-infected juniper plants is compulsory in Austria and Switzerland (1, 9). An epiphytotic of the disease was reported in 1948 at Lausanne, Switzerland (2). Deckenbach (8) considered it to be one of the most dangerous diseases of pear in the Crimea, destroying the whole crop in certain districts.

The damage is confined to the leaves, fruit, twigs, and branches of pear. The rust does not seem to cause any appreciable injury to its alternate host, the junipers.

In Europe, direct control of the trellis rust has been obtained by spraying the pear trees repeatedly each spring with Bordeaux mixture. If spraying for the rust is necessary in North America, undoubtedly ferric dimethyl dithiocarbamate (ferbam) or manganese ethylene bisdithiocarbamate (maneb) will be preferred, as these fungicides have proved effective against other rust species on apple (7). Indirect control has been obtained by eradication of junipers or rust-infected juniper branches within a radius of at least 1000 feet from the pears. Eradication of junipers is considered to be the only practical method to control trellis rust (2, 10), particularly in areas where the rust has been recently introduced.

DISCUSSION

Trellis rust became established in Victoria presumably from infected Juniperus sabina vars. imported from Europe in or prior to the year of 1945. Most of the diseased junipers apparently grew in one garden; unfortunately these were destroyed in the spring of 1960 before they could be examined by a mycologist. Most of the specimens at hand were collected from junipers of a neighbor's garden. For reasons as yet undetermined the rust does not seem to have spread farther than 1000 feet since it was introduced. This is surprising, since (a) the age of some rust cankers, as well as reports of property owners, indicate that some infections of juniper actually have taken place after the juniper was imported from Europe; (b) junipers, particularly J. sabina vars., are widely used as ornamentals in Greater Victoria, often in the vicinity of pear trees; and (c) under prevailing climatic conditions, junipers within hundreds of miles from the rusted pear trees might well have become infected during the past 15 years.

Although susceptibility of hosts in only the Sabina section of Juniperus has been proven experimentally (10), Kern (12) believes that there is "much evidence that it is also on the Oxycedrus section."

Conners (7) suspects physiological specialization in the quince rust (Gymnosporangium clavipes Cke. & Pk.), which, he implies, may have taken place in the trellis rust, also:

> "What I think is significant is the fact that the rust is known on species of Juniperus in the two sections, Oxycedrus and Sabina. It may have been fortunate that the rust was imported on J. sabina, for I suspect from my observations on G. clavipes that the rust is specialized to the two sections of the genus (in fact, morphologically they are not quite the same). Ottawa is about the northern limit of J. virginiana, where it is host only to G. globosum, although G. clavipes is abundant on J. communis var. depressa at Ottawa and is not difficult to find on J. virginiana some 50 miles to the southwest. The distribution of G. clavipes and G. globosum in the Arboretum is according to which section of the genus the host belongs."

The potential danger of the trellis rust to pear orchards in North America is difficult to estimate until more is known about the susceptibility of native junipers to this European parasite.

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DOWNY MILDEW OF RICE IN ARKANSAS[1]

G. E. Templeton, T. H. Johnston, and S. E. Henry[2]

In August 1958, one malformed panicle of the variety Century Patna 231 rice (CI 8993) was found at the Rice Branch Experiment Station, Stuttgart, Arkansas (Fig. 1A). The tissue of this panicle was sectioned and was found to contain oospores (Fig. 1B) of the same size range and

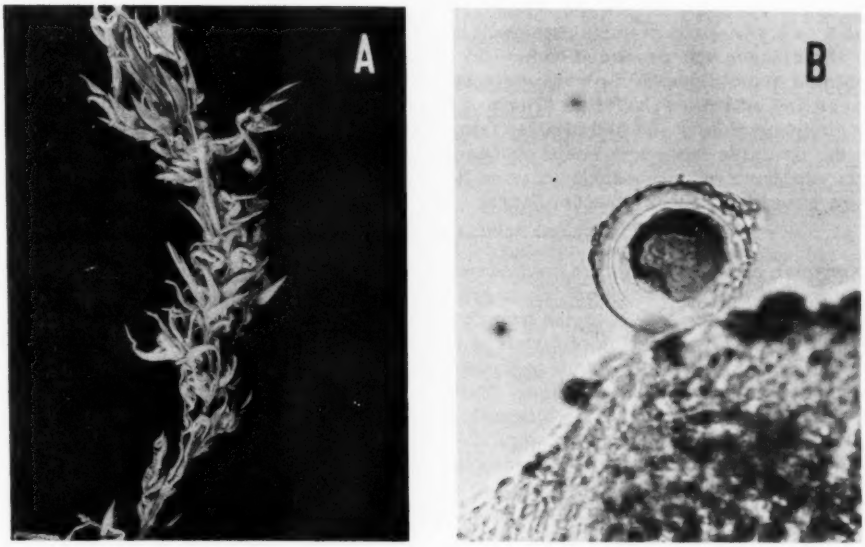

FIGURE 1. A -- Symptoms of downy mildew on rice.
B -- Oospore from diseased tissue.

description as those reported for Sclerophthora macrospora (Sacc.) Thirum., C. G. Shaw & Naras on small grains and grasses in Arkansas[3]. As far as we were able to determine, this organism has never been reported on rice in the Western Hemisphere. Its potential seriousness prompted a search for similarly malformed panicles during 1959 and 1960. Limited surveys were made in the rice area of the State in fields where conditions were similar to those believed favorable for expression of the disease. No other diseased panicles were found.

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[3]Dale, J. L., and G. E. Templeton. 1960. Downy mildew of small grains and grasses in Arkansas. Plant Disease Reptr. 44: 206-207.

AN UNDESCRIBED BLIGHT DISEASE OF ARIZONA CYPRESS[1]

Charles L. Wilson[2]

Summary

Cercospora thujina Plakidas was found associated with a blight disease of Arizona cypress (Cupressus arizonica). The disease starts in the lower, inner branches and progresses upward and outward. Needles turn bronze and become necrotic. Small trees 2 to 3 years old may be killed within a year.

The disease was produced on healthy Arizona cypress cuttings by isolates of C. thujina from diseased Arizona cypress trees. Isolates of C. thujina from Arizona cypress and oriental arborvitae (Thuja orientalis), a known host, were indistinguishable morphologically and had similar temperature and nutrient requirements for growth. Isolates from both hosts produced abortive spores in culture. Isolates of C. thujina from oriental arborvitae and Arizona cypress produced a blighting of the foliage of oriental arborvitae seedlings.

Nurserymen and homeowners have been experiencing difficulty in establishing plantings of Arizona cypress (Cupressus arizonica) in Arkansas. The difficulty is due primarily to a blight disease that starts in the lower, inner branches and progresses upward and outward until the tree may be killed. Examination of diseased branches from 3-year-old plantings near Hope, Arkansas and at the University Farm in Fayetteville revealed that Cercospora thujina was consistently associated with the disease. Material collected in Macon, Georgia also had fruiting of C. thujina associated with the blighted foliage[3]. Exosporium glomerulosum (Sacc.) Hoehn was also found associated with some diseased tissue at the University Farm. Both C. thujina and E. glomerulosum have been reported to cause blight diseases of other coniferous trees (3, 4) but neither have been reported to occur on Arizona cypress[4].

Phomopsis juniperovora Hahn has long been recognized as an important pathogen of coniferous seedlings (2). It has been considered the main cause of a blight of Arizona cypress. In 1945 Plakidas (4) described a blighting fungus of oriental arborvitae (Thuja orientalis) and Italian cypress (Cupressus sempervirens)which he named C. thujina. He also found this fungus sporulating on Arizona cypress[4]. Plakidas (4) indicated in his description of C. thujina that the conidia were faintly echinulated. Chupp (1) in his monographic study felt that C. thujina was not a true Cercospora and should, because of the echinulations, be placed in the genus Heterosporium. Kelman, et al. (3) recently found E. glomerulosum associated with a blight disease of Juniperus virginiana in North Carolina.

This paper presents a comparative study of C. thujina on Arizona cypress and oriental arborvitae. The possible involvement of E. glomerulosum in the blighting of Arizona cypress foliage was not investigated.

MATERIALS AND METHODS

All isolations of C. thujina were made on a malt extract-yeast extract agar medium (10 g malt extract and 2 g yeast extract). Cultures were obtained by touching a small piece of agar to conidia produced on diseased foliage and placing the seeded agar on the medium. This method proved very effective for obtaining pure cultures. Inoculum was grown in liquid shake cultures on the malt extract-yeast extract medium. C. thujina fragments in liquid culture and produces numerous pellets of growth. The growth was filtered out of the medium and macerated in water in a Waring Blendor. Baker variety of oriental arborvitae was used in the inoculations because it was reported (3) to be highly susceptible. Terminal cuttings of Arizona cypress, 6 to 11 inches long, were dipped in the inoculum and the checks were dipped in distilled water. They were then placed erect in flats of sand and a polyethylene tent was constructed

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The author is indebted to Dr. A. G. Plakidas for examining material and giving helpful suggestions concerning this investigation.

[3]The material from Georgia was kindly supplied by Dr. A. A. Foster.

[4]Dr. A. G. Plakidas stated in correspondence that he has observed C. thujina sporulating on Arizona cypress foliage in Louisiana.

around the flats. The cuttings were kept covered in a humid environment for 48 hours after inoculation. Potted oriental arborvitae seedlings about a foot tall were sprayed with the inoculum and placed in the field. The checks were left unsprayed.

An isolate of C. thujina from Arizona cypress and one from oriental arborvitae were grown at different temperatures on three natural media: Difco malt agar, Difco prune agar, Difco potato-dextrose agar. The media were prepared according the manufacturer's directions. Two Petri dishes implanted with each isolate were incubated at temperatures ranging from 4° to 40° C at 4-degree intervals. The radial growth of the colonies at the various temperatures was recorded at the end of 25 days. Because of the extremely slow rate of growth of this organism, it was not practical to record growth at shorter intervals.

EXPERIMENTAL RESULTS

Symptoms of the Disease: This disease causes a progressive necrosis of the needles and stem tissue and a tree eventually may be killed. It starts in the lower, inner branches and progresses upward and outward. As it progresses, the tips of the needles usually become necrotic, then the remainder of the needle turns bronze. Later, affected needles and branches become reddish-brown and may be shed. Small trees can be killed within a year, or in some cases the disease appears to remain static on the lower branches. In a 3-year-old planting observed during the 1958 growing season at the University Farm, most of the necrosis occurred from mid-September to late November. The disease causes desiccation of needles and stem tissue in advance of necrosis. Terminal branches which are affected at their base become flaccid. This probably results from infection of the stem. In the planting at the University Farm, the disease has not been particularly severe in any one area, but is random in its occurrence.

Comparisons of C. thujina from Arizona Cypress and Oriental Arborvitae: The fungus on blighted Arizona cypress was found to be indistinguishable morphologically from C. thujina as described by Plakidas (4), both in nature and in culture. The length and width of the conidia and conidiophores in nature fell within the same general range (Table 1). The number of septations of the spores from both sources also were similar.

Table 1. Measurements of Cercospora thujina in nature.

Source	Conidia	Conidiophores	Septations
Arizona cypress	range	range	range
(50 measured)	30-70 x 4.0 x 7.0μ	26-56 x 4.0-6.0μ	2-8
	average	average	average
	47.8 x 6.1μ	46 x 4.3μ	4
Arborvitae	range	range	range
(50 measured)	36-82 x 4.4-8.4μ	24-68 x 4.0 x 6.6μ	0-8
	average	average	average
	51.8 x 6.4μ	49 x 4.6μ	3

The cultural characteristics of C. thujina from Arizona cypress and oriental arborvitae on agar and in liquid culture were indistinguishable. C. thujina grows very slowly on an agar medium. The maximum radial growth obtained after 25 days was 12 mm. Growth is more rapid in liquid shake culture and this method was more satisfactory for obtaining inoculum. On agar, C. thujina produces two distinct types of hyphae. Hyaline, aerial hyphae are subtended by darkly pigmented, larger hyphae which form a stromatic-like growth. The dark hyphae often fragment to form individual cells. The width, form, and pigmentation of these two types of hyphae were compared in cultures from Arizona cypress and oriental arborvitae and found to be indistinguishable. Cultures from oriental arborvitae and Arizona cypress both produced occasional spores on malt agar. These spores were not normal in form or in number of septations, but they did correspond to normal conidia in that they were echinulated and of a similar color. They are produced terminally on hyphae that are pigmented.

The growth of two isolates of C. thujina, one from Arizona cypress and one from oriental arborvitae, was compared on three natural media. Both isolates: 1) grew best on the malt agar medium, 2) had the same temperature range (16° to 32° C) for growth on all three media, and 3) had an optimum for growth of 24° on the malt agar and prune agar, and an optimum of 28° on the potato-dextrose agar medium (Table 2).

Table 2. Comparative growth of C. thujina on three natural media after 25 days.

Medium	Source of isolate	Maximum radial growth (cm)	Optimum temperature for growth (°C)	Temperature range for growth (°C)
Difco malt agar	Arizona cypress	12.0	24	16-32
	arborvitae	9.7	24	16-32
Difco prune agar	Arizona cypress	9.0	24	16-32
	arborvitae	8.5	24	16-32
Difco potato-dextrose agar	Arizona cypress	9.2	28	16-32
	arborvitae	8.5	28	16-32

Table 3. Inoculations with Cercospora thujina.

Method and date of inoculation	Source of isolation	Host inoculated	Reaction and date
Cuttings in sand culture 2-2-59	Arizona cypress	Arizona cypress	Inoculated cuttings (30) blighted, fruiting present, fungus reisolated 3-15-59, and uninoculated cuttings (30) still green and turgid 4-15-59
Potted plants placed in the field 5-2-59	Arizona cypress arborvitae	arborvitae	Both isolates gave similar reaction. Inoculated plants (4) blighted, fruiting present, fungus reisolated 7-4-59, and uninoculated plants (4) still green and turgid 8-5-59

Inoculations: Isolates of C. thujina from both Arizona cypress and oriental arborvitae produced a blight disease when placed on healthy Arizona cypress cuttings (Table 3). Symptoms of the disease were similar to those found in nature. Infection occurred when isolates from either Arizona cypress or arborvitae were placed on arborvitae seedlings. Symptoms produced by isolates from either host were indistinguishable (Table 3). C. thujina was found sporulating on most of the needles that had been recently killed and the fungus was reisolated. The cultures obtained were indistinguishable from other isolates of C. thujina.

Sporulation on Diseased Tissue: Branches from diseased trees were examined weekly in 1959 from the first of March until sporulation of C. thujina was found. Sporulation of C. thujina was not detected until May 30 in the planting at the University Farm at Fayetteville. Sporulation of C. thujina is olivaceous and is not easily detected on needles that still have some green color. It is generally found on the proximal side of the needle. C. thujina and E. glomerulosum were the only fungi found sporulating on tissue showing incipient stages of the disease.

An Alternaria sp., Pestalozzia sp., Phoma sp., and Periconia sp. were found on branches that had been necrotic for some time. The Alternaria sp. was most prevalent and imparted a sooty appearance to dead branches. The pathogenicity of these organisms was not tested. Because they were found only on tissue that was necrotic, it is thought that they were probably secondary invaders.

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A NEW KERNEL ROT DISEASE OF CORN IN WISCONSIN[1]

Paul E. Hoppe[2]

An extremely severe kernel rot disease that affected every ear of corn inbred W59M was observed at two widely separated locations in Wisconsin in 1960. This susceptible line was developed at the Branch Experiment Station, Spooner, Wisconsin and is used as the pistillate parent in making the single hybrid (W59M x W117).

The disease occurred in a 5-acre single-cross production field near Black River Falls and at Arlington. These locations, respectively, are about 135 and 250 miles southeast of Spooner. At Arlington, the susceptible inbred was planted in close proximity to inbred W117 and the F_1 (W59M x W117), each in 40-hill plots. Because of total disease, the 5-acre production field was abandoned.

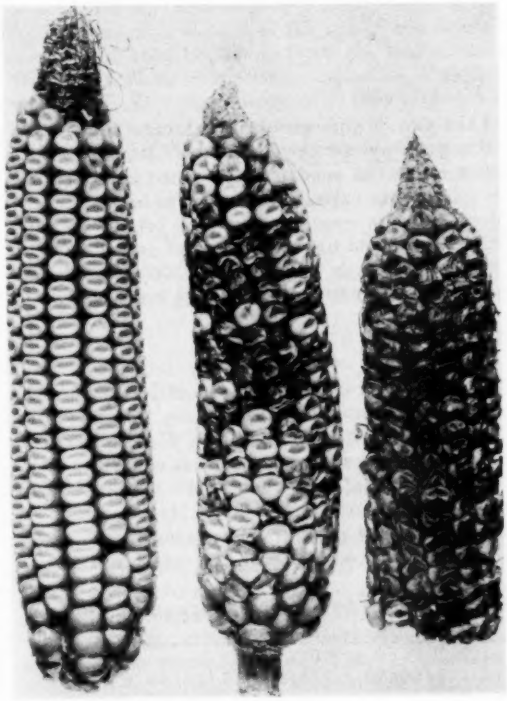

FIGURE 1. Healthy and diseased ears of corn inbred 59M illustrating kernel rot that affected every ear of this line at two widely separated locations in Wisconsin in 1960. The healthy ear (left) was grown at a third location.

In a second crossing field, also near Black River Falls but where better soil drainage prevailed, the kernel rot failed to develop. At Spooner, the disease has been observed in this line in former years, but never to a degree to cause special concern.

The kernel rot was not observed in the F_1 (W59M x W117) grown essentially alongside the inbreds at Arlington. This indicates that susceptibility is carried as a genetic recessive in the susceptible parent.

Disease Symptoms: Healthy and diseased ears are illustrated in Figure 1. The majority of the ears in the above-mentioned seed production field showed over 75% of the kernels diseased, the number varying from as few as 25 on some ears to all kernels rotted on others. When the condition first was observed, the affected kernels were light brown or tan in color and seemed to contain excessive moisture, as though they were in a stage of soft rot disintegration. The kernels were covered with a sticky exudate that adhered to the inner husks when these were pulled back. After the ears were collected and dried, the diseased kernels became shriveled and somewhat darker in color. Little solid material remained within the pericarp of most diseased seeds.

Isolations and Soft Rot Tests: Isolations from diseased kernels of 30 ears selected at random yielded a variety of bacterial colonies. Each of the isolates was tested in Petri plates for soft rot potentials on onion and potato tissues. Some appeared nonpathogenic, but the majority caused soft rot in one or both hosts in varying degrees of virulence.

This previously undescribed kernel rot disease that was so devastating at Black River Falls represents the first reported instance of a disease causing 100% loss in a Wisconsin cornfield. A few of the bacterial cultures that proved highly virulent in the soft rot tests were saved for field inoculations in 1961.

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[1]Cooperative investigations of the Crops Research Division, Agricultural Research Service, United States Department of Agriculture and the Wisconsin Agricultural Experiment Station.

[2]Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture and the Wisconsin Agricultural Experiment Station.

RESULTS OF PRELIMINARY TESTS WITH PHENACRIDANE CHLORIDE FOR THE CONTROL OF FIREBLIGHT OF APPLE AND BACTERIAL SPOT OF PEACH

J. W. Bushong and Dwight Powell[1]

Summary

Seven spray applications of phenacridane chloride (9-(p-n-hexyloxyphenyl)-10-methyl-acridinium chloride)[2] used at 100, 200, and 400 ppm at 8 to 12 day intervals effectively controlled natural twig blight infection of fireblight of apples.

The amount and intensity of bacterial spot on peach leaves was markedly reduced by phenacridane chloride at 100, 200, and 400 ppm. The trees were sprayed at intervals of 8 to 16 days during the season prior to an artificial inoculation of the leaves with a bacterial suspension.

INTRODUCTION

Smale and Mitchell (1) were the first to report the use of phenacridane chloride and closely related compounds as effective against the plant pathogen Phytophthora phaseoli Thaxt.

To our knowledge no reports have been published about the sensitivity of plant pathogenic bacteria to phenacridane chloride. Unpublished in vitro tests (2) have shown it to be highly effective against Erwinia amylovora (Burr.) Winslow et al. in concentrations as low as 10 ppm. Since there is no satisfactory spray for control of fireblight and bacterial spot of peach Xanthomonas pruni (E. F. Sm.) Dows. experiments were undertaken to determine the possible use of phenacridane chloride as a control measure for these diseases and the results are reported here.

METHODS

Fireblight Experiment: Phenacridane chloride was used at concentrations of 100, 200 and 400 ppm. DuPont spreader-sticker, at the rate of 2 ounces/100 gallons of water, was added to all of the sprays including a water control. The spray apparatus consisted of a 2-gallon pressure tank. Two 9-year-old Jonathan apple trees were chosen for each treatment and 30 to 35 succulent twigs per tree were selected at random. The terminal 12 inches of the twigs were sprayed thoroughly. The twigs were sprayed at 8 to 12 day intervals: May 19, May 31, June 10, June 18, June 26, July 5, and July 14. Data were recorded at intervals during the course of the experiment by counting the number of naturally occurring twig blights (Tables 1 and 2).

Table 1. Dates observed and number of twigs blighted by E. amylovora.

Treatment (ppm)	5/31/60	6/10/60	6/18/60	6/26/60	7/5/60	7/14/60
100	0	0	0	2	4	4
200	0	0	0	0	4	6
400	0	0	0	0	3	5
Control	0	0	9	15	26	27

Table 2. Percentage of twigs blighted and percentage of control of fireblight.

Treatment (ppm)	Total number twigs	Total number of twigs blighted	% fireblight	% control
100	62	4	6.4	84.1
200	62	6	9.7	75.9
400	62	5	8.1	79.9
Control	67	27	40.3	----

[1]Research Assistant and Professor of Plant Pathology, respectively, Department of Plant Pathology, University of Illinois, Urbana, Illinois.

[2]Material supplied under code EP-166 by Research Department, Morton Chemical Company, Woodstock, Illinois.

Bacterial Spot Experiment: Ten twigs on each of four 7-year-old Elberta peach trees were selected at random for each treatment. Eight spray concentrations of 100, 200 and 400 ppm of phenacridane chloride plus the DuPont spreader-sticker, and eight sprays of water plus sticker were used as treatments. The spray was applied on the following dates: June 2, June 10, June 18, June 26, July 5, July 14, August 6 and August 22. The terminal 15 to 18 inches of the twigs and leaves were thoroughly sprayed on both upper and lower surfaces using the same method as in the fireblight experiment. Natural infections did not occur, therefore, artificial inoculation of the sprayed trees was necessary. The inoculum was prepared in the following manner: Bacterial cells of X. pruni were grown on modified Emerson's agar slants for 48 hours at 30°C. The culture tubes were then filled with sterile deionized water and a sterile chromium wire loop was scraped across the agar to dislodge the cells from the medium. The bacterial suspensions were diluted by pouring them into one large flask containing sterile deionized water. The number of bacterial cells in the final suspension was 8.1×10^6 per ml as determined by the standard plating and counting technique (3). The bacterial suspension was applied under pressure to the upper and lower leaf surfaces of all treated twigs. The spray nozzle was held 12 inches from the leaf surface and a direct stream was allowed to hit the leaves. The inoculations were made August 25, 3 days after the last spray, and data were recorded September 5. The percentage of leaves infected and the intensity of infection are shown in Table 3.

Table 3. Percentage of peach leaves infected with bacterial spot using three different intensities of infection: 0 spots per leaf, 1-9 spots per leaf, and > 10 spots per leaf.

Treatment (ppm)	% leaves infected and grouped according to intensity of infection		
	0 spots per leaf	1-9 spots per leaf	>10 spots per leaf
100	70	26	4
200	95	5	5
400	87	5	8
Control	26	25	49

CONCLUSIONS

Although these experiments should be considered preliminary, the data definitely indicate a high amount of activity in preventing infection by phenacridane chloride at all concentrations. Moderately severe bacterial spot infections occurred at the 100 ppm treatment, but the disease was greatly reduced by the 200 and 400 ppm concentrations of phenacridane chloride. Since no phytotoxicity was observed from any concentration it is probable that higher concentrations would be more effective in controlling fireblight. Higher concentrations, however, might cause a phytotoxic condition to develop. It is interesting to note that in addition to reducing the amount of fireblight, symptoms of developing infections were delayed by 1 to 2 weeks on the treated twigs. In these experiments the material acted as a protectant rather than as an eradicant.

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CAVITY SPOT DISEASE OF CARROT AND PARSNIP ROOTS[1]

Emil F. Guba, Robert E. Young, and Tadao Ui[2]

Abstract

Cavity spot of carrot roots is a physiologic disease. It appears rarely on parsnip roots, and its history in the United States dates back to 1936. In Massachusetts, Waltham Hicolor carrot shows an inherent weakness to the trouble. Cavities appear in the cortex below the epidermis. The covering tissue collapses and the area is discolored and rotted by bacteria and molds. Cork layers of cells develop, and healing and camouflaging of the lesions, if sterile, occur. The trouble has a genetic background and in Massachusetts it became important in crop culture about the time the Waltham Hicolor carrot was introduced. Its prevalence is greater in growing seasons marked by high rainfall and culture under some form of malnutrition, as can be implied by serious occurrences of the trouble on roots grown in beds leached with water prior to planting, or excessively watered during culture, or culture on barren land.

HISTORICAL

In October 1956, a peculiar spotting of parsnip roots was found on an Essex County, Massachusetts vegetable farm (Fig. 1). The discolored lesions were actually sunken craters or cavities in the surface tissue of the roots. The color of the spots was between pink and brown, and some decay was present. Bacteria, and less frequently the fungus Fusarium, were found in the lesions. Similar lesions were noted in roots of the Waltham Hicolor carrot, and to a lesser extent among roots of other carrot varieties (Fig. 2). There were no foliage symptoms of disease coincident with the root trouble.

FIGURE 1. Cavity spot lesions on parsnip root, Hollow Crown variety, Andover, Mass., October 10, 1956.

FIGURE 2. Cavity spot lesions on carrot root, Hicolor variety, Lincoln, Mass., September 1958.

Ramsey (6) described apparently similar root lesions on California carrots in 1936. The blemishes were unusual and in many instances there was some decay. Fusarium was consistently isolated. The trouble was again described by Ramsey and Wiant (7) as blemishes invaded by Fusarium resulting in the formation of shallow corky or scabby areas. They reported, "The lesions are of chief importance as a blemish of the roots although occasionally a shallow decay develops that seldom penetrates deeper than one-eighth of an inch." A similar trouble is seen occasionally on California carrots in consumer vegetable outlets in Massachusetts today.

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A scab disease of carrot roots was described by Ark and Gardner (1) in 1944 in California and attributed to the bacterium Phytomonas carotae (Kendrick) Bergey et al. Earlier, Kendrick (4) in California described a bacterial blight of carrot foliage caused by a new species of bacterium, Pseudomonas carotae Kendrick. Ark and Gardner attributed both the root and foliage phases of the disease to the same bacterial pathogen and on the basis of substantial proof. Similar circumstances were reported by Brown (2) with isolates from diseased carrots from Arizona and south Texas. The description and illustrations of the root lesions by Brown, p. 6, Pl. 3, are similar to the carrot root disease in Massachusetts.

Kenknight and Blodgett (5) reported that the pathogen Xanthomonas carotae (Kendrick) Dowson was a major foliage disease of the carrot seed crop in Idaho in the period 1943-1945 but that there was no root phase of the disease. Also, Watson (8) and letter of October 2, 1958, reported that the root phase of the pathogen had never been observed in Idaho. Further compilations of the literature on the disease were reported by Whitaker, et al. (9).

In 1952, Grogan and Kendrick (3) reported that carrot root scab in California bears a variety of fungus and bacterial organisms, none of which is pathogenic. The reddish brown lesions between the lines of lateral roots are associated with oil ducts and oil-filled, unbroken blisters on the roots. The trouble can be serious on one root and absent on an adjoining root. Grogan (letter Dec. 20, 1958) stated that root scab is influenced by fertilizer levels and temperature. Some varieties show more severe disease than others. Cross sections of the blister-like areas indicated that they are closely associated with the rupture of oil ducts. Their work does not support previous reports that the disease is caused by the bacterium Xanthomonas carotae (Kendrick) Dowson.

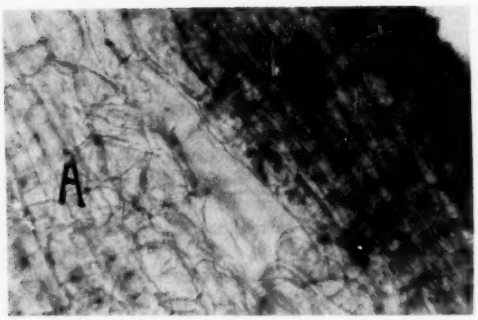
ROOT CAVITY SPOT

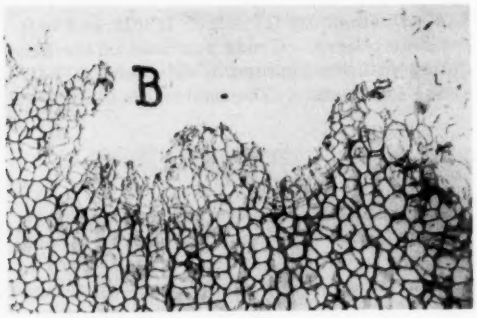
The cavity spots in carrot and parsnip roots in Massachusetts hardly come within the form or concept of scab lesions (Figs. 1, 2). The cavities with small openings at the surface, suggest injuries by the maggot of the carrot rust fly, and they are mistaken for maggot injury by growers. The cavities often heal as the root grows, leaving a shallow clean laterally elongated scar. Usually tissue around the cavity is infected and discolored. The cavities are first formed under the epidermis and are hidden from view (Fig. 3A). The covering tissue ruptures and the lesions assume the form of a sunken cavity in the surface tissue of the root. The cavities have an irregular lobed or ragged outline, and they are as much as 3 to 4 mm deep (Fig. 3B, C). Some roots show an abundance of lesions, while other adjoining them may be free or show but a small number of them. The openings or apertures are 0.2 x 0.5 to 1.5 x 4mm in diameter. The cavities are always deeper radially and wider laterally than the opening. The lesions are scattered and they are usually more numerous on the upper than the lower part of the roots. If they are numerous their distribution is irregular; otherwise they are often arranged vertically on the root.

The margin of the cavities is usually lined with layers of cork cells in response to injury and infection (Figs. 3B, C; 4). Often the callous cells develop only at the base of the cavity (Fig. 3B). Bacteria and mycelium are present in the margin of the cavity, and the presence of bacteria is more frequent than mycelium (Figs. 4A, B). Penetration of the tissue expands the area of decay and discoloration. Some cavities appear to originate from oil ducts, but no clear proof of this was found. In paraffin microtome sections abnormally developed oil ducts were not found.

The Model variety of parsnip grown in greenhouse steam sterilized beds showed more cavity spot disease than Hollow Crown and All America. With carrots there was considerable variation in the prevalence of the trouble among different lots of Waltham Hicolor. Hutchinson and Gold Pak showed as much as Waltham Hicolor with one exception. In a later planting in the same bed, Danvers Half Long and Waltham Hicolor were most seriously affected, and all varieties showed much disease. In a comparative varietal planting in the field in 1959 there were marked differences in the prevalence of the disease among different selections of Waltham Hicolor, indicating the genetic aspects of the trouble. Some lots showed a little; other varieties, with the exception of Hutchinson, were practically free. In 1959 Waltham Hicolor in succession and triplicate plantings in the field showed more disease than Hutchinson. In commercial culture the disease is important on Waltham Hicolor.

Artificial cultures of tissue from cavity lesions yielded bacteria and molds. Isolations of bacteria and the fungus Fusarium were frequent. Most of the tissue cultures were sterile. Of 52 tissue cultures from the margins of cavity lesions in carrots, 26 were sterile. Of 25 cultures from submarginal tissue of lesions in carrots, 20 were sterile. In another set of 50 tis-





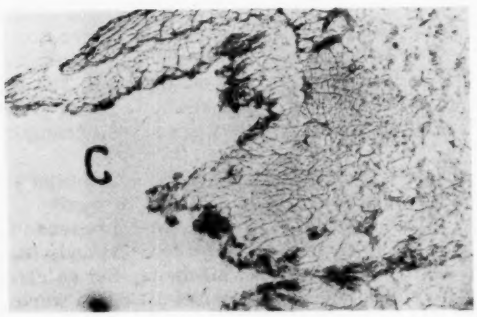
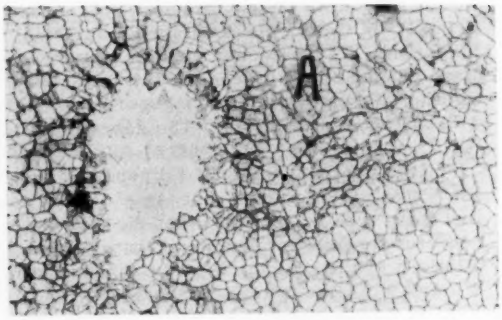
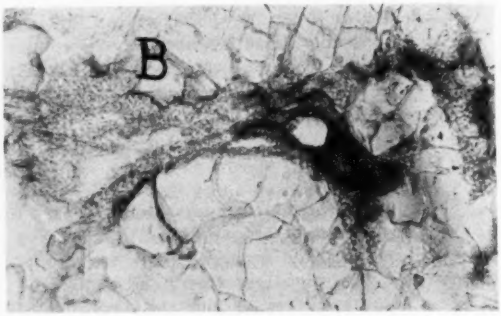


FIGURE 3. Microtome sections of cavity spot lesions. A -- Initiation of cavity spot lesion in cortex of carrot root under superficial white mottled area. B -- Cross section showing development of layers of cork cells about the base of the cavity. C -- Cross section at opening showing character of infected tissue and the irregular margin of the cavity.





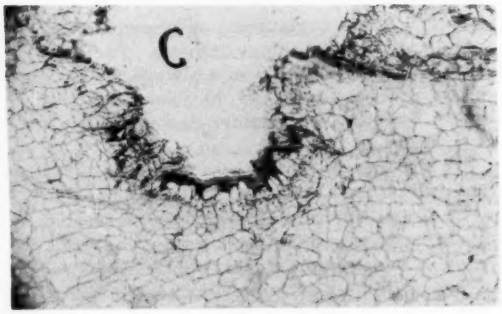


FIGURE 4. Microtome sections of cavity spot lesions. A -- Cross section of indentation in margin of cavity. Mycelium and conidia of Fusarium and bacteria are apparent. B -- Bacteria penetrating tissue beyond the margin of cavity and causing decay. C -- Margin of cavity infected by bacteria and molds and host cell growth responding to injury and infection.

sue cultures from the sub surface of white mottled areas in carrots, 38 were negative. Of 50 cultures from submarginal tissue of lesions in parsnip roots, 19 were negative. The positive cultures comprised bacteria and molds of Fusarium, Penicillium, Cephalosporium, Alternaria, Cylindrocarpon and Mycelia sterilia. Some of them caused decay following inoculation of sterile carrot slices; others were innocuous. The fact that only a fraction of the tissue cultures yielded organisms, and only a fraction of the isolates produced decay, supports the opinion of the physiologic character of the disease and the secondary role of the organisms.

Carrots grown in steam sterilized soil in the greenhouse showed a high incidence of lesions. The spots were free of decay. Roots from unsterilized soil showed discolored lesions and decay.

In greenhouse culture there was more disease on roots from beds kept on the wet side by frequent watering. It was greatest in beds leached with water before planting. The least incidence appeared in wet plots fertilized with 8-16-16 fertilizer before planting. Under dry culture the greatest incidence of disease occurred where the plot was leached with water before planting. The results suggest malnutrition as a factor in the development of the trouble.

Significant variation in the incidence of the disease in field culture appeared on roots from different planting sites and from among successive planting dates. Barren land never in cultivation and lacking fertility and humus showed the greatest incidence of root cavity spot. There was more of the disease generally in the roots in plantings of carrots on May 27 than on May 13 in 1959.

CONCLUSIONS

Grogan and Kendrick (3) reported that root scab lesions result indirectly from the interaction of a certain genotype and climatic factors peculiar to the central coastal section of California. The genetic aspects of the trouble are recognized. Cavity root lesions are not too apparent if they are not discolored by infection. The use of irrigation water would require caution, to discourage infection. Proper soil fertility for vigor and as an aid to healing and camouflaging the lesions is also implied. Thus, good soil fertility and drainage are recommended. The disease represents a worthy subject of fundamental study in plant physiology.

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CONTROLLING MILDEW ON APPLES AT WENATCHEE, WASHINGTON IN 1960[1]

Roderick Sprague[2]

Summary

The newer materials that warrant further testing for control of apple powdery mildew are: Phytopabst and DY-Q-Plex-1 and perhaps 06K-50W. In the doubtful class are: Ortho methyl mercury-8 and Rayonier MS-2029 mostly because of injury to the host or, as in the case of the mercury, because of poisonous properties. Dyrene and nickel sulfate were discarded. Under severe mildew conditions dodine (Cyprex), a scab spray, was weak against the mildew.

The search for more effective, safer and possibly less expensive fungicides to control powdery mildew (Podosphaera leucotricha) in eastern Washington was continued in 1960 (Tables 1 and 2).

Powdery mildew on Jonathan apples in the mildew plot at Wenatchee was rampant in 1960, one of the worst years since 1948. A fairly heavy carryover of mildew was noted at the first count on April 6 at early pre-pink. The first spray on April 7 with such recognized materials as Karathane 25% W. P. was effective. However, from pink spray time, April 13, to about full bloom on April 24, rain and wind were prevalent during cold, unfavorable growing weather. The condition continued past April 24 with increasing winds. There was a period of 15 days from April 24 until May 9 when it was impossible to apply a spray. When a fourth spray was finally applied on May 9, mildew in the unsprayed checks averaged 54.8% leaf surface injury, while control with all materials had not shown much improvement since the first spray (Table 2). It was necessary, therefore, to add a fifth spray of most materials to get practical control.

In calculating percentage loss shown in Table 2, use was made of a previously reported method wherein the percentage of clusters or terminals of leaves mildewed were first counted and then classified by the severity of mildew coverage into class 1 (severe) to 5 (very light). Finally the figure for the percentage of cluster was divided by the class number. If, for instance, the checks show 54.6% clusters mildewed and the injury is severe (factor 1): 54.6 ÷ 1 = 54.6 calculated percentage loss. If one has 30% of the clusters on Karathane-sprayed trees mildewed but the loss is moderately light with a factor of 3, take 30 divided by 3 or 10% calculated loss in surface area. This rule of thumb system is accurate enough to permit large-scale sampling of the rather extensive local plots.

The tests were made in a solid block of 12-year-old Black Jonathans with several dozen unsprayed trees scattered through the area. The five-tree replications were hand sprayed.

Results again indicated that Karathane 25% W. P. at the 3/4-pound rate gave adequate control. Under the adverse conditions of 1960, five sprays were necessary. The 50% emulsion of Karathane at the 6-ounce rate worked as well as the 25% W. P. A sticker was used with W. P. and none with emulsion. A standard treatment using Polysulfide Compound at four pounds on April 7, 3 1/2 pounds on April 14 and 2 1/2 pounds on April 24 disclosed need of a fourth spray before the mildew counts were over. During a more average season this three-spray schedule is generally practical. It is a rare grower who applies more than three mildew sprays of anything, in fact, not too many reach the three-spray level.

Two formulations of Phytopabst, AL 379 and AL 307 (Phytoactin), were received late and applied on mature leaves. The antibiotic gives good control in mid-season on mature leaves in the field. In the greenhouse on juvenile leaves of pears its effectiveness is less. Until large enough samples are available to test on young leaves in the field in early season, the value of this material for mildew control is uncertain.

Rayonier MS-2029 is a nickel carrying powder with the odor of fresh sawdust. It caused severe leaf injury and extreme fruit damage at 4 pounds/100 gallons. Mildew was reduced, mainly by shock treatment, followed by leaf cast. Strangely enough, these trees recovered and at mid-summer looked healthy except for the fruit.

Naugatuck 06K-50W was received late after greenhouse tests had shown some promise with laboratory samples. The one test was inconclusive.

A finely divided copper hydroxide complex, DY-Q-Plex-1, was received late. On mature leaves it appeared to dry down mildew and surprisingly caused no injury. Since coppers are notoriously unreliable in north-central Washington, this material needs testing on tender young leaves in the early season. The 1/2-pound rate appeared to be adequate. It should be tested against fire blight using dosages as low as 4 ounces/100 gallons.

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[1]Scientific Paper 2031, Washington Agricultural Experiment Stations, Pullman, Washington, Contribution from Department of Plant Pathology. Project No. 1164.

[2]Plant Pathologist, Washington State University, Tree Fruit Experiment Station, Wenatchee, Washington.

Table 1. Table of spray chemicals used.

Active ingredient	Formulation	Formulator
2,4 dinitro-6(2-octyl) phenyl crotonate	Karathane WD (25% W. P.)	Rohm & Haas Co., Philadelphia, Pennsylvania
2,4 dinitro-6(2-octyl) phenyl crotonate	Karathane Liq. Conc. (50% liquid technical Karathane)	Rohm & Haas Co., Philadelphia, Pennsylvania
Sodium polysulfide (84%) and sodium thiosulfate (8%)	Polysulfide Compound	Niagara Chemical Division, Food Machinery & Chem. Corp., Richmond, California
Methyl mercury oxyquinolinate	Ortho methyl mercury-8 (5.7% Hg)	California Spray-Chemical Corp.
Nickel sulfate chelated on to cellulose	MS-2029	Rayonier Inc., Shelton, Washington
A polypeptide antibiotic derived from Streptomyces	Phytopabst AL 379 and also AL 307	Pabst Brewing Co., Milwaukee, Wisconsin
2,4-dichloro-6-(o-chloroanilino-s-triazine	Dyrene	Chemagro Corp., Kansas City, Missouri
2-amino-3 chloro-1,4 naphthoquinone	06K-50W	Naugatuck Chemicals, Bethany, Connecticut
Nickel sulfate	Nickel sulfate	Allied Chemical Corp.
Dodecylguanidine acetate	Cyprex (65%)	American Cyanamid Co.
Copper hydroxide complex	DY-Q-Plex-1	Henry Bower Chemical Manufacturing Co., Philadelphia, Pennsylvania

Table 2. Results of spraying 5-tree blocks of 12-year-old Black Jonathan apples with a spray gun (1 x strength material) at Wenatchee, Washington, 1960.

Material	Rate/100 gallons	Dates applied	Calculated percentage loss on: Apr. 6	Apr. 13	Apr. 24	May 10	May 18	June 1	June 20	July 12
Karathane W.P. 25%	3/4 lb	April 7, 14, 24 May 9, 19	27.3	8.6	7.8	9.6	6.6	7.3	10.5	13.2
Karathane 50% l. emul.	6 ozs	April 7, 14, 24 May 9, 19	20.8	9.0	8.9	8.0	9.1	10.3	13.0	11.0
Polysulfide Compound	4 lbs, 3 1/2 lbs, 2 1/2 lbs	April 7, 14, 24	22.9	10.1	6.2	8.9	10.5	16.4	19.3	21.0
Ortho methyl mercury 8	1/2 pt	April 7, 14, 24 May 9	17.1	15.7	11.5	17.2	13.8	19.6	10.0	13.4
Unsprayed checks	--	--	36.5	41.4	43.5	54.8	60.2	64.3	64.7	70.0
Rayonier MS-2029	4 lbs, 2 lbs	April 7, 14, 24 4 lbs; May 9, 2 lbs	34.5	25.3	8.6	8.1	5.7	9.9	11.6	13.8
Phytopabst AL 379	1 qt	May 9, 19; June 1			43.3		19.6	17.6	12.6	21.3
Phytopabst AL 307	1 qt	May 9, 19; June 1			31.4		13.8	10.1	12.8	15.3
Dyrene	2 lbs	June 1						50.4	32.4	34.2
Naugatuck 06K-50W	1 lb	June 10						39.4	18.2	27.3
Nickel sulfate	3/4 lb	June 21							56.4	37.0
Cyprex (dodine)	3/4 lb	May 9			35.2		26.9	41.4	44.1	42.2
DY-Q-Plex-1	1/2 lb	July 6; August 5							50.4	23.7

VERTICILLIUM WILT OF SAUCER MAGNOLIA, MAGNOLIA SOULANGEANA

Lawrence R. Schreiber, Claude Fordyce, Jr., and R. J. Green, Jr.[1]

In August 1960 a saucer magnolia, Magnolia soulangeana, was examined that was severely wilted. The tree measured 11 inches d.b.h. and was approximately 60 years old. Symptoms included inrolling and necrosis of the leaves and extensive greenish-brown vascular discoloration. The cut ends of the larger branches showed additional areas of vascular discoloration deep in the xylem (Fig. 1), suggesting that the tree had been infected much earlier.

Isolations from the discolored sapwood regularly yielded Verticillium albo-atrum Reinke & Berth. The tree was kept under observation and within 14 days after symptoms were first observed complete defoliation had occurred and the dormant buds were dead.

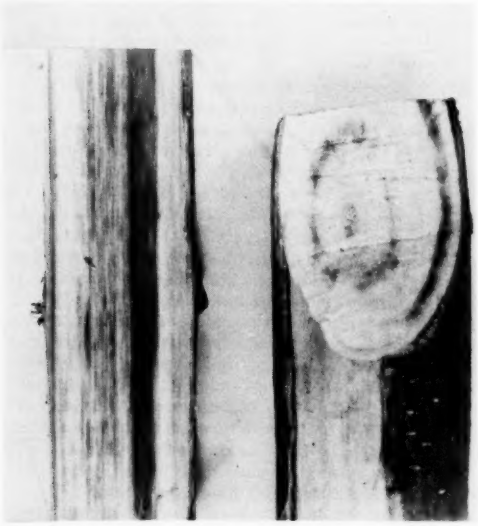

FIGURE 1. Vascular discoloration of saucer magnolia associated with Verticillium wilt disease. Note the presence of discoloration in the older wood.

FIGURE 2. Symptoms of Verticillium wilt on saucer magnolia artificially inoculated with V. albo-atrum (after 6 weeks).

Greenhouse studies established the pathogenicity of V. albo-atrum isolates. Several small plants of M. soulangeana were inoculated by root-dipping into a suspension of mycelium-microsclerotia of the pathogen. The trees were potted and held at 20° to 24° C for 6 weeks. After 4 weeks the inoculated plants wilted and after 6 weeks exhibited extensive leaf necrosis and vascular discoloration (Fig. 2). Verticillium albo-atrum was reisolated from the sapwood of inoculated plants.

Cross-inoculation studies were made using the Bonny Best variety of tomato. Tomato seedlings were inoculated by root dipping and observed for 6 weeks. No symptoms appeared at the end of this period and attempts to reisolate the pathogen were unsuccessful.

This is the first report of Verticillium wilt disease of a member of the genus Magnolia, although the disease has been reported in the genus Liriodendron of the family Magnoliaceae.

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A DETERMINATION OF NECROTIC-RING-SPOT VIRUS IN MICHIGAN PEACH ORCHARDS AND NURSERY STOCK

Donald Cation[1]

Abstract

Necrotic-ring-spot virus, previously considered present in negligible amounts in Michigan peach trees, was found prevalent in nursery trees and young peach plantings. Limited indexings from older peach plantings were negative. The evidence from indexing and 20 years' observation indicates that Prunus ring-spot virus does not spread from peach to peach or from cherry to peach in Michigan. Prunus ring spot infections are apparently introduced in nursery trees through infected seedling rootstock and infected scion wood.

Naturally occurring infections of necrotic-ring-spot virus have not been reported from Michigan peach orchards. Many inoculations of the past 20 years' testing of Michigan peach trees for viruses and virus-like disorders have not indicated necrotic-ring-spot virus. To the contrary, seedling peach trees grown from Michigan cannery pits have responded as expected when inoculated with known necrotic ring spot isolates by showing typical delayed foliation, necrotic and chlorotic ring spots, and smaller leaves, with twig and limb dieback. These symptoms are followed by apparent recovery although the virus remains in the tree. Only a virus-free tree will show symptoms following inoculation. An inoculated seedling tree reacting with symptoms shows indirect evidence that it was originally free from the virus. The little or no indication of necrotic-ring-spot virus in Michigan peach orchards has led to a complacent attitude in regard to this disease and it was not considered necessary to include the disease in the peach tree certification program. The finding by Millikan (2) of necrotic ring spot in midwestern nursery stock and in 15% of Lovell seedlings prompted a re-analysis of Michigan peach nursery stock and orchard plantings.

MATERIALS AND METHODS

A total of 400 peach trees were indexed during the winter on Montmorency cherry in the greenhouse or during August on Shirofugan flowering cherry in the field. The indexings included 160 trees, consisting of 10 trees each of currently popular peach varieties obtained from two Michigan nurseries and one Arkansas nursery, 62 orchard trees selected as a possible source of budwood, 60 trees from a 2-year-old commercial orchard, 20 trees from a 25-year-old orchard in Oceana County, 10 trees from an old abandoned orchard in Berrien County, 88 uninoculated trees from the virus test orchard at East Lansing, 101 Rio Oso Gem trees propagated in 1959 at East Lansing and 10 Red Leaf trees being grown as a seed source.

Indexing of Nursery Trees: The indexing results for nursery trees are given in Table 1. More trees from nurseries A and C showed necrotic-ring-spot infection than from nursery B. It may be significant that the trees from nurseries A and C were culls or small-sized compared with the normal-sized trees provided by nursery B. Half the 10 Rio Oso Gem trees individually tested from nursery A were infected. Results with 30 other Rio Oso Gem trees (not included in Table 1) indicated that they were infected in about the same proportion. The propagation wood for the 40 Rio Oso Gem trees was obtained from an outside source by the nursery and not from the nursery orchard.

The data from this determination do not indicate whether the seedling stock or the budwood source was responsible for the large amount of necrotic-ring-spot virus in nursery A, but the lack of infection in three varieties points to contaminated budwood as a contributing factor with the other three.

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Table 1. Necrotic-ring-spot infections in 10-tree varietal samples from three nurseries.

Variety	Nursery A	Nursery B	Nursery C[a]
Elberta	0	0	0
Halehaven	5	0	1
Richhaven	0	0	-
Sunhaven	3	0	-
Rio Oso Gem	5	-	-
Amber Gem	0	-	-
Red Skin	-	3	-
Red Bird	-	-	3
J. H. Hale	-	-	3
Golden Jubilee	-	-	1

[a] Located in Arkansas.

Indexing Trees from a Budwood Source Orchard: Five varieties in nursery A orchard were indexed to establish a source of virus-free budwood for 1960 propagation and to follow through with further study. Of interest is the high incidence of necrotic ring spot found in Sunhaven, a relatively new variety. This and other indexing results are presented in Table 2.

Table 2. Necrotic-ring-spot indexing results for trees from several peach plantings.

Variety	Nursery A budwood orchard		2-year-old commercial orchard		Oceana and Berrien co. orchards		Virus-test orchard E. Lansing	
	+	-	+	-	+	-	+	-
Elberta	.....		1	9	0	10	0	28
Sunhaven	6	4	2	8	.....		0	19
Richhaven	1	9	0	10	.....		0	32
Halehaven	.....		.....		0	10	.....	
Red Skin	1	9	1	9	.....		.....	
Redhaven	0	8	1	9	.....		.....	
Kalhaven	0	10	.....		.....		.....	
Rio Oso Gem	1	13	1	9	.....		0	101
Undetermined	.....		.....		0	10	.....	

Indexing Trees from a 2-year-old Orchard: One or more infected trees were found in five of six varieties in a 2-year-old orchard located near South Haven. The age of these trees and the evident lack of tree-to-tree spread in Michigan clearly indicates that necrotic-ring-spot virus was in the nursery stock planted in this orchard.

Indexing Trees from Old Orchards: The 25-year-old orchard in Oceana County is in an area in which there is a high incidence of necrotic-ring-spot and cherry-yellows virus in cherries. The orchard was immediately adjacent to infected cherry trees on the south and east. Ten consecutive Halehaven trees from the south row and 10 consecutive Elberta trees from the east row were free of necrotic-ring-spot virus in the indexing test.

Ten consecutive trees from an old abandoned orchard in Berrien County, also in an area with a high incidence of necrotic ring spot in cherry trees, were likewise free of necrotic-ring-spot virus.

Indexing Uninoculated Trees in a Virus-test Orchard: Twenty-eight Elberta, 19 Sunhaven and 32 Richhaven 4-year-old uninoculated trees and 10 6-year-old Red-Leaf peach trees in the virus-test orchard at East Lansing gave a negative index for necrotic ring spot. The Elberta, Sunhaven and Richhaven trees had been propagated on seedlings grown from Michigan cannery pits. The Led-Leaf peach trees were purchased from Michigan nurseries and are being grown for a seed source. These trees are interspersed with various virus-inoculated peach trees and are immediately adjacent to cherry trees infected with necrotic-ring-spot and sour-cherry-yellows virus. The trees receive relatively few (3 to 4) insecticidal sprays each season. Necrotic-ring-spot virus is actively spreading from cherry to cherry in this block but has not been observed to spread from peach to peach or from cherry to peach.

Indexing Locally Grown Nursery Trees: One hundred and one Rio Oso Gem nursery trees

grown on seedlings from Michigan cannery Elberta pits were indexed and found negative. This result, together with past experience, indicates that Halehaven and Elberta pits from Michigan canneries carry a low incidence of necrotic-ring-spot virus.

DISCUSSION

The limited sampling from older trees and from trees grown on seedlings from Michigan cannery pits corroborates past experience that necrotic ring spot has been a negligible factor in Michigan peach plantings. Most Michigan peach orchards are relatively near cherries known to be contaminated with necrotic-ring-spot virus. It would be expected that the virus could not gain entrance to established peach trees in Michigan without expression of shock symptoms, thus assuring eventual detection. Such shock symptoms of necrotic ring spot or "shock and stunt" symptoms of sour cherry yellows have not been observed in Michigan peach orchards.

The large amount of necrotic-ring-spot virus indicated in some peach nursery stock may be of relatively recent occurrence. The virus could be transmitted either through seed or budwood sources. Millikan's (2) determination that 15% of Lovell seedlings were infected perhaps approached the maximum expected from that source if the findings of Cochran (1) and Wagnon, et al. (3) are considered. These workers found from 1 to 11% of the seeds from a ring-spot-infected tree carried the virus, the amount of infection varying with the variety. If virus-free budwood is used for propagation on Lovell seedlings, then presumably a maximum of 15% of the nursery trees would be infected. This amount might actually be less since there is possibly some mortality when disease-free buds are inserted on infected seedlings.

If virus-infected buds were inserted on Lovell seedlings the 15% infected seedlings should produce a full stand of good appearing trees, whereas the remaining 85% virus-free seedlings would become infected and produce less than a full stand of somewhat less than full-sized trees. Such was indicated in an experience of several years ago when imported Dixie Red buds showed a very poor "take" and virus-shock symptoms were evident on the budded seedlings.

There has been no study to show that the necrotic-ring-spot virus impairs the size and yield of orchard peach trees except for trees in shock. However, there is good evidence that infection with the necrotic-spot strain of the ring-spot virus results in a lower grade of nursery trees and that the other strains in general reduce the size of peach nursery trees by 15% (3).

It may not be possible to eliminate necrotic-ring-spot virus from peach stock entirely until a clean source of seed is generally available to the nursery trade. However, it appears feasible to reduce the disease to a minimum by the relatively easy chore of indexing the few budwood source trees required for each nursery. Eliminating necrotic-ring-spot virus from peach nursery stock appears to offer more benefit to the nurseryman than to the grower. The result should be a more uniform stand of larger sized trees.

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FUNGICIDAL TESTS FOR CONTROL OF FUSARIUM PATCH DISEASE OF TURF[1]

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Abstract

Fusarium patch is the most troublesome disease of golf putting turf in western Washington. Organic mercurials have given faster and better control of this disease than have inorganic mercury or cadmium compounds, thiram, and several other types of fungicides. Cadmium compounds, however, were usually less phytotoxic and turf treated with certain types was usually more dense than that treated with organic mercurials. Effective control has been obtained with 3/4 ounce of phenylmercury acetate (PMA as a 10% solution) in 10 gallons water/1000 square feet applied every 2 weeks. Phytotoxicity of PMA was overcome by adding 1/8 to 1/4 pound/1000 square feet of available nitrogen from calcium or ammonium nitrate sources. The most promising approach, which combines the best features of both types of materials, is an alternating schedule of phenylmercury acetate and cadmium chloride (as Caddy at 1 ounce).

INTRODUCTION

Fusarium patch, caused by Fusarium nivale (Fr.) Ces., is the most serious disease of golf putting-green turf and the second most serious disease of lawn-type turf in western Washington. It occurs every month of the year but is usually most serious in late spring and particularly in the fall (1).

In 1955 Meiners (2) reported on the importance and control of the disease in eastern Washington. Studies were initiated in western Washington in 1956 in response to a request from the Northwest Turf Association. This article summarizes the general results obtained in the 11 field experiments to date.

MATERIALS AND METHODS

Preliminary tests for disease control and phytotoxicity with certain materials were made on turf in a lathhouse (once) or a plastic greenhouse (twice). The results of these tests are not reported except for a few pertinent points. The field tests were run on an experimental green at the Western Washington Experiment Station or on greens at the following golf clubs: Allenmore Golf Club in Tacoma, Broadmoor Golf and Country Club in Seattle, and Overlake Golf and Country Club in Bellevue. Plots were 5 x 5 feet, replicated five or more times. The plots were randomized on a basis of known or expected natural disease development. Materials were applied as sprays except for one commercial dry formulation of phenyl mercuric acetate. In certain tests, the rates of mercury and cadmium compounds were adjusted to the amounts of actual mercury in PMAS and cadmium in Caddy. The solutions were applied at the rate of 10 gallons/1000 square feet every 2 weeks, unless otherwise stated. Pressure in the sprayer was kept constant through use of compressed air.

Whenever possible, and particularly in the later tests, data were obtained on the three factors: disease control, color, and density. The number of spots was used as the criterion of disease control. However, with the poorer materials, the spots were not only more numerous, but were usually two to three times larger in diameter and took longer to heal. Therefore, a count of 1000 versus 500 spots represents much more than twice as much trouble. Color was rated from 1 to 10 with the highest number representing the greenest grass. The color was an indication of interaction of phytotoxicity, disease control, and nitrogen fertilization. Turf density was rated from 1 to 10. It usually, but not always, represented the interaction between disease control and phytotoxicity. The latter varied considerably, being most severe with succulent grass and high temperatures.

The general performances of most materials included in field tests are rated in Table 1. If a compound failed to control the disease or burned or thinned the grass, it is listed with one or more minus signs; if the color and density are comparable to the check, but not appreciably better, a + is used; more than one + indicates the degree of superiority over the untreated area. Each rating is a composite one based on all experiments with each material.

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Table 1. Most important materials tested and general results obtained on control of Fusarium patch disease of putting-green turf in western Washington, 1956-1959.

Fungicides	Composition and source	Rate (ounces/1000 sq. ft.)	Grams of Hg or Cd/1000 sq. ft.	Results[a]			
				No. times tested	Disease control	Degree of phytotoxicity	Density
	MERCURIALS						
Caloclor	Mercurous chloride (60%) + mercuric chloride (30%) (M)	0.50	10.4	1			+++
		1.00	20.7	3	++	+	+++
		2.00	41.4	1	++	--	+++
Calocure	Hg_2Cl_2 (30%) + $HgCl_2$ (15%) + $(NH_4)_2SO_4$ (25%) + ferrous sulfate (25%) + gum acacia (5%) (M)	2.00	21.0	5	+	+	++
		3.00	31.5	3	++	+	++
Ceresan 200	Ethyl mercury acetate (1.3%) + ethyl mercury 2,3-dihydroxy propyl mercaptide (6%) (D)	0.5	0.67	2	-	+	+
		1.0	1.34	1	+	--	+
Elcide 70	(Thimerosal) sodium ethyl mercury thiosalicylate (6%) (L)	0.75	0.67	1		+	+
Emmi	N-ethylmercuri-1,2,3,6-tetrahydro-3,6-endomethano-3,4,5,6,7,7-hexachloro-phthalimide (10.3%) (V)	1.30	1.31	1		+	-
Liquiphene	Phenyl mercuric acetate (33.3%) (Vi)	0.25	1.48	1		+	+
Meta Sol P-6	Phenyl mercury propionate (10%) (Me)	0.75	1.27	1	+++	---	++
Panogen Turf Spray	Methyl mercury dicyandiamide (2.2%)(Mo)	2.00	0.89	1		+	+
PMAS	Phenyl mercury acetate (10%) (Cl)	0.50	0.89	2	+++	-	+++
		0.75	1.34	8	++++	--	++
		1.00	1.78	3	++++	----	++
		2.00	3.56	1	++++	----	+
Puraspra	Phenyl mercury triethanol ammonium lactate (1.66%) (MP)	9.00	1.73	1	++++	----	+
Scutl V-4a	Phenyl mercury salts (Commercial Scutl) (Sc)	19.3	?	3	++++	---	-
Special Semesan	Hydroxymercuri chlorophenol (21.5%) + hydroxymercuri cresol (3.8%) (D)	2.00	8.3	1	++	+	++
		4.00	16.6	2	+++	+	++
Tag	Phenyl mercury acetate (10%) (C)	0.75	1.34	1	++++	--	++
Tersan OM	Hydroxymercury chlorophenol (10%) + thiram (45%) (D)	4.00	6.59	1	-	+	++
Thimer	Phenyl mercury acetate (3%) + thiram (75%) (Cl)	3.00	1.53	1	+	+	+++
	CADMIUMS						
Caddy	Cadmium chloride (20.1%) (Cl)	0.50	1.82	1		+	+
		0.75	2.73	1		+	+
		1.00	3.64	4	+++	+	+++
Cadminate	Cadmium succinate (60%) (M)	0.50	4.19	1	++	+	++
		1.00	8.38	1	++	+	++
Kromad	Cd sebacate (5%) + potassium chromate (5%) + malachite green (1%) + thiram (15%) + auramine (0.5%) + iron sulfate (16%) + urea (54.5%) + wetting agent (2%) (M)	2.00	1.02	2	+	+	+
		3.00	1.53	2		+	++
		4.00	2.04	1	++	+	++
Ortho 1567	$CdCO_3$ (3.25% Cd) + captan (60%) + Hi-12 (15%) + BHC (1.5%) (C)	4.00	3.70	3	++	+	+++
Ortho 1741	$CdCO_3$ (3.26% Cd) + Phaltan (60%) + Hi-12 (15%) + BHC (1.5%) (C)	4.00	3.70	2	++	++	+++
Ortho 1742	$CdCl_2$ (3.26% Cd) + captan (60%) + Hi-12 (15%) + BHC (1.5%) (C)	4.00	3.70	2	++	+	+++
Puraturf 177	Phenyl amino cadmium dilactate (20%) (G)	1.5	2.56	1		+	++

Sources: C=Calif. Spray, Cl=Cleary, D=duPont, G=Gallowhur, L=Lilly, M=Mallinckrodt, Me=Metalsalts, Mo = Morton, MP=Miller Prod., Sc=Scott, V=Velsicol, Vi=Vineland.

[a] - to ---- indicates decreasing disease control and turf density, and increasing phytotoxicity. + to ++++ indicates increasing benefit. Lack of + or - indicates no reading taken or possible.

RESULTS

Mercury Compounds

The organic mercurials have proven much more effective than inorganic mercurials in controlling Fusarium patch in western Washington. For instance, at the peak of disease development in one experiment, there were 845 spots in the untreated area as compared with 304 in the Caloclor (at 1 ounce) per 125 square feet. There were only 36 in the PMAS (at 3/4 ounce) area, in spite of the fact that the Caloclor contained over 15 times as much mercury as PMAS. Increasing the dosage of Caloclor to 2 ounces reduced the disease count to 71 in this test, but increasing the interval from 2 to 3 weeks (to reduce costs) decreased the control (327 spots).

However, some data and observations indicate that on certain greens and under certain conditions the inorganic mercuries give better control than at other times. In one test Calocure at 3 ounces gave almost as good disease reduction as PMAS at 1/2 ounce (a lower-than-normal rate) and produced a much thicker turf. In a later test, Calocure at 3 ounces provided very poor control in the spring (725 spots versus 120 for PMAS at 3/4 ounce and 1085 for the untreated) but gave somewhat better control in late fall (172 spots versus 0 for PMAS and 731 for the check).

It was thought possible that the inorganic mercury compounds might have a longer residual action than organic compounds, thereby making use of the material more economical. This was not found to be the case. When the timing interval was increased from 2 to 3 and to 4 weeks, the disease control became progressively inferior. Thus, in one of the 1957 experiments, Caloclor at 1 ounce was applied at 2-, 3-, and 4-week intervals beginning September 4. Numbers of diseased spots were approximately uniform (300 ± per 125 square feet) at start of the experiment. Counts of diseased spots throughout the season are listed in Table 2.

Table 2. Effect of timing on control with PMAS and Caloclor (Broadmoor).

Treatment (starting 9/4/57)	Total number diseased spots for all 5 reps. (125 square feet)					% grass 11/14	General rating 12/30 (40=best)
	Sept. 20	Sept. 30	Oct. 16	Nov. 14	Total		
Untreated	605	1025	990	1185	3805	58	21
PMAS[a] at 2 weeks	23	25	21	44	113	93	40
3	55	50	24	21	150	95	40
4	28	424	406	485	1343	76	32
Caloclor[b] at 2 weeks	178	358	483	442	1461	76	36
3	365	524	477	501	1867	77	32
4	401	648	530	749	2328	69	27

[a] at 3/4 ounce. [b] at 1 ounce.

Based on all results, heavier applications of Calocure possibly might provide reasonably good Fusarium control; however, cost would be an important item. Caloclor is somewhat cheaper than Calocure and somewhat more effective in controlling Fusarium; however, it has been more phytotoxic under certain conditions. The difference in response is probably caused by inclusion of nitrogen in the Calocure formulation.

Not all organic mercurials were equally effective. Thus, phenylmercury acetate brought the disease under control in the field somewhat more rapidly and more effectively than did a hydroxymercury compound. For example, spots sprayed with the latter compound (as Special Semesan at 4 ounces) had 173 spots versus 37 for PMAS at 1 ounce and 525 for the untreated in spite of a mercury content that was nine times higher than that in PMAS. Phenylmercury propionate was less effective than phenylmercury acetate at similar concentrations of actual mercury. Most of the other phenylmercury formulations appeared promising. In fact, it is probable that satisfactory control could be obtained with most organic mercurials, if sufficiently high rates were economical and not phytotoxic.

The various formulations of phenylmercurials seemed to vary more in phytotoxicity than they did in fungicidal action. At the same approximate rate of mercury, phenylmercury propionate was somewhat more phytotoxic than phenylmercury acetate. Even different formulations of the latter varied in phytotoxicity. All four formulations of phenylmercurials (Liquiphene, PMAS, PMA Sol. #10, and Tag), used at rates of 1 ounce (10% active) or stronger, caused severe injury. The dry phenylmercury (Scutl) produced considerable injury to putting

turf when applied every 2 weeks, less when applied every 4 weeks, and very little when used on lawn turf.

Commercial mixtures of organic mercurials with tetramethylthiuram disulfide (thiram) (Tersan OM and Thimer) gave poor results at recommended rates, even though the mercury content was comparable with, or higher than, that of PMAS at 3/4 ounce.

The density of turf in various treatments was usually an interaction between disease control and phytotoxicity. In general, the organic mercurials appeared to have a thinning action on turf in western Washington when disease was not a factor. Under similar conditions the inorganic mercurials sometimes produced a thicker turf than the organic mercurials.

Since PMAS had controlled the disease effectively in our preliminary experiments and since it was generally available, it was used for subsequent studies on timing, rates, and other factors. The following discussion, therefore, deals with PMAS unless otherwise noted. Results with PMAS would likely apply to other similar formulations of phenylmercury acetate.

PMAS - Rates: PMAS has been tested 73 times in 14 greenhouse and field experiments since 1956. Included with these tests were some on rates which varied from 1/2 ounce to 3 ounces. The higher rates used at first gave excellent control but considerable phytotoxicity. The disease control in such "burned" grass lasted for several weeks after cessation of application. This could have been caused by elimination of the fungus, induced hardiness of the plants, or some other physiological factor, such as an apparent decreased production of water of guttation. Despite the excellent disease control, the high rates were generally considered impractical, so lower rates were tested.

In one experiment in 1958, applications of PMAS at 1/2, 3/4 and 1 ounce were compared. All three rates effectively controlled the disease. The 1/2-ounce rate gave good control in this test but in other tests it did not usually produce as rapid control as the 3/4-ounce rate. When grass was growing rapidly, the diseased spots usually filled in within 2 to 3 weeks after treatment with 3/4 ounce of PMAS but not until after 4 to 5 weeks for the 1/2-ounce rate (at 2-week spraying intervals). The 1-ounce rate gave good control but was phytotoxic. Therefore, since 1/2 ounce appeared to be a "marginal" rate, the 3/4-ounce level was selected for most subsequent work and for general recommendation. Since the 3/4-ounce and even the 1/2-ounce rate still gave some burning at times, efforts were made to counteract this in later experiments.

PMAS - Timing: Most efficient golf course superintendents prefer to schedule as many operations as possible for the same day of each week. An early test indicated that a 14-day interval was apparently as satisfactory as the 10-day interval often recommended for turf disease control. Next, it appeared desirable to determine if satisfactory control could be obtained with applications every 3 or 4 weeks instead of every 2 weeks. In a test in 1957, applications were started on September 4 and continued at 2-, 3-, and 4-week intervals using PMAS at 3/4 ounce. Results are shown in Table 2.

Control was slightly better from the 2-week than the 3-week interval and definitely better than the 4-week. Other tests indicated that with a regular spraying program, a 3-week interval was usually adequate but somewhat "marginal." Therefore, a bi-weekly schedule was adopted for future work and for general recommendation. However, the phytotoxicity was generally reduced and the density of turf increased where 3- and 4-week intervals were used instead of 2 weeks in these studies.

An alternating 3-week schedule of PMAS at 3/4 ounce and Caloclor at 1 ounce was also included in this experiment. The number of diseased spots tended to drop after each application of PMAS but started increasing after each application of Caloclor. Similar, but less striking, results were obtained in 1958 using an alternating schedule of PMAS at 3/4 ounce and Calocure at 3 ounces. The same trends were noted in other experiments using Caloclor at 1 ounce in a 2-, 3-, and 4-week timing study (Table 2) and Calocure at 3 ounces in a 2- versus a 3-week schedule; better control was obtained at the shorter interval.

PMAS - Amount of Water for Dilution: Recommendations for turf disease control often suggest the use of fungicides in either 5 or 10 gallons of water/1000 square feet. A dilution of 10 gallons was used in most of our tests, but the two amounts of water were compared in a greenhouse test in 1959 with PMAS at 3/4 ounce. Although the same amount of mercury was applied in each case/square foot, the plots in the 5-gallon series were burned much more severely than in the 10-gallon series. Since similar results had been obtained with Caloclor at 1 ounce in 1957 and since PMAS at 3/4 ounce was a marginal rate, insofar as burning was concerned, the 10-gallon dilution was adopted as standard.

PMAS +Iron or Neburon: Ferrous sulfate is often sprayed on turf for "greening" purposes. If it could be incorporated with the fungicide, a considerable saving in labor would result. Tests with PMAS at 3/4 ounce/1000 square feet plus ferrous sulfate at 1/2, 1, and 2 ounces and

sequestrene iron at 1 ounce indicated that the fungicidal effectiveness was not appreciably altered but that in periods of stress the combination was more phytotoxic than the PMAS alone. The addition of certain nitrogenous materials to the combination of PMAS and iron reduced this burning.

Turf on plots treated continuously with mercury compounds often became thin during cloudy weather, with a subsequent development of algae. Since 1-n-butyl-3-(3, 4-dichlorophenyl)-1-methylurea (neburon) had successfully controlled the algae on putting greens when applied separately, it was tried at 1/2 ounce/1000 square feet with PMAS. The combination usually resulted in poorer disease control and considerably more phytotoxicity. Although less burning resulted from lower rates of neburon, particularly when nitrogen was included, the combination is considered hazardous.

PMAS + Nitrogenous Compounds: Preliminary tests indicated that the addition of certain nitrogenous compounds to PMAS could reduce the phytotoxicity of the compound. Consequently, research was expanded along this line in 1958 and 1959. It was soon apparent that nitrogen at rates above 1/2 pound (actual N)/1000 square feet often increased the phytotoxicity and decreased control, so rates of 1/8, 1/4, and 1/2 pound were used in subsequent studies. Urea, even at 1/2 pound, markedly increased the burn and decreased control, so work with it was discontinued. Ammonium hydroxide appeared more promising than other forms of nitrogen in one test with Mersolite W (a different PMA product) and should probably be retested. However, we subsequently directed most of our efforts to the nitrate sources.

One-half and 1/4 pounds of available N/1000 square feet from Ca, K, and NH_4 nitrates were used with PMAS at 3/4 ounce in the spring of 1959 on the Allenmore Golf Course. The weather turned warm and dry and disease development ceased, but results indicated that calcium nitrate at 1/2 pound increased the phytotoxicity; ammonium nitrate at 1/2 and 1/4 pounds slightly decreased it; and potassium nitrate had little effect under the conditions at that time.

Based upon the preceding results, a similar experiment was initiated on a putting green at the Broadmoor Golf Club on September 3, 1959. Applications were continued at bi-weekly intervals until December 23, except for one 3-week interval in October following a time when the green was accidentally aerified and heavily sanded by the club. Seven replications of 5 x 5 feet plots were used in this test. After an initial outbreak in early September, the disease intensity generally declined in early October and then began increasing toward the end of that month.

Results are shown in Table 3 for the PMAS-nitrogen combinations and most other fungicides tested at the same time. The PMAS + 1/2 pound N combinations soon began burning the turf so these rates of nitrogen were changed to 1/8 pound on October 1. However, the effect of the higher rates of nitrogen apparently persisted for a long time, as evidenced by color and disease development. Since high rates of nitrogen had increased disease in previous tests, and since the higher (1/4 pound) rate of calcium nitrate in this test showed somewhat more disease than the lower rate, we believe the results in plots with the 1/2 pound initial rate of potassium and ammonium nitrates may indicate a prolonged effect from the two heavy applications of nitrogen at the start of the experiment. The delayed and prolonged increase in number of diseased spots may also indicate an effect primarily on the host, rather than on the fungus. Differences in disease control between the different types of nitrates at the 1/4-pound rate were minor. Ammonium nitrate seemed slightly superior to the calcium and potassium types in turf density and color, but slightly inferior in disease control.

Another test was initiated on October 26, 1959 on the Overlake Golf Course. Calcium, potassium and ammonium nitrates were all used at a rate of 1/8 pound of actual nitrogen with PMAS at 3/4 ounce. Applications were continued at bi-weekly intervals until December 23. All three nitrogen types effectively eliminated the phytotoxicity of PMAS, greened up the grass, and increased the density of the turf. Disease development was too inadequate to note any differences between treatments.

The data obtained have shown that the phytotoxicity of PMAS can effectively be reduced or eliminated without appreciably affecting disease control by adding low amounts of nitrate or ammonium nitrate nitrogen to the solution. The level of nitrogen to be used should be adjusted according to turf condition, rainfall and temperature; but probably should not exceed 1/8 pound/1000 square feet of available nitrogen in western Washington. The amount of nitrogen added may also have to be adjusted to the type of fungicidal compound since certain formulations already contain some nitrogen. All available evidence indicates that high rates of nitrogen increase susceptibility of grass to attack by Fusarium nivale, and that at least one type (urea) may be worse than others in this regard. The addition of nitrogen with fungicides that either were ineffective or that gave only fair control was usually worse than no treatment at all.

Table 3. Results of fungicide application on disease control, color and density of putting-green turf. Broadmoor, 1959.

Fungicide	Rate of fungicide/ 1000 sq.ft. (ounces)	Fertilizer	Rate of N/ 1000 sq. ft. (pounds)	No. diseased spots[a]/175 sq. ft. (7 reps.) 1959					Total through 12-29	No. diseased spots/175 sq. ft. 1960		Average rating[b]	
				9-21	10-9	11-5	11-18	12-29		2-24	5-20	color	density
None				472	119	1390	1075	1220	4276	575	229	7.2	5.3
PMAS	3/4			64	5	275	205	230	779	113	89	5.6	6.1
PMAS	do.	$CaNO_3$	1/8	100	4	345	180	260	889	108	83	7.0	6.6
do.	do.	$CaNO_3$	1/4	14	2	408	300	440	1164	180	145	7.3	5.9
do.	do.	KNO_3	1/4	78	4	350	290	340	1062	129	86	7.1	6.4
do.	do.	KNO_3	1/2→1/8	67	4	665	470	570	1776	241	129	7.0	5.9
do.	do.	NH_4NO_3	1/4	107	4	454	270	365	1200	83	73	7.4	6.5
do.	do.	NH_4NO_3	1/2→1/8	88	1	645	530	545	1809	178	140	6.7	5.8
Tag	do.			62	6	277	160	195	700	65	81	5.9	6.2
Puraspra	9			52	7	275	110	240	684	138	51	4.8	5.2
Tersan OM	4			322	16	1340	1095	1115	3888	605	326	7.3	5.9
Caddy	1			0	0	299	185	150	634	78	11	7.1	7.1
PMAS alt. Caddy	3/4 or 1			24	0	191	126	110	451	48	24	6.8	7.0
Met Sol P-6	3/4			44	4	438	335	410	1231	223	71	5.5	5.9
Ortho 1567	4			1	0	770	750	535	2056	202	215	7.5	7.2
Ortho 1741	do.			10	0	900	755	670	2335	365	114	7.9	6.8
Ortho 1742	do.			3	0	775	640	585	2003	239	290	7.5	7.2
Scutl F-4a - at 2 wks. - 4 1/2 (sp)[c]				57	1	213	125	180	576	108	71	5.2	4.7
Scutl F-4a - at 4 wks. - 4 1/2 (sp)[c]				153	0	294	150	255	832	92	61	6.1	6.6

[a]The number of diseased spots was used as the criterion of disease control. However, the spots were two to four times as large in the less effective treatments and took longer to heal.
[b]Total of individual plot ratings 1 to 10, with 10 being best color and perfect stand.
[c]Scott's Spreader at 4 1/2 opening.

The residual effect of various treatments in certain tests was noticeable. Thus, in the Broadmoor experiment started in 1959, differences were still present on April 20 in 1960, four months after applications ceased. The relative ratings remained about the same. The untreated plots and those sprayed with poorer materials continued to exhibit the most disease; while the plots with treatments that had given the best disease control in the fall continued to have a low incidence of diseased spots (Table 3). These results occurred despite the fact that inocula must have been distributed by temporary flooding during heavy rains and by mowers which operated at least weekly during the mild winter. The fungicides may have accumulated sufficiently

in the soil to provide a prolonged kill of introduced spores and mycelial fragments. Also it is probable that the normal incubation period of the Fusarium is much longer than heretofore suspected, particularly during cool weather. In any case, the results definitely show the benefit from a regular schedule of treatments.

Cadmium Compounds

The cadmium compounds, as a group, have given poorer disease control than have the mercurials, but the most effective cadmiums have usually produced a denser turf, except during severe epiphytotics. Certain cadmium compounds, such as cadmium dimethyl dithiocarbamate (in greenhouse tests) either failed to control the fungus or at times seemed to increase it, perhaps through the elimination of beneficial organisms. Cadmium chloride (as Caddy) has given the best over-all results of all the cadmium compounds thoroughly tested to date. Caddy and an alternating schedule of Caddy and PMAS resulted in better control of the disease in the 1959 Broadmoor test than any other treatment (Table 3). The relative performance of Caddy was somewhat better than usual in this 1959 test, but it has kept the disease reasonably well under control even during severe epiphytotics. If we take into consideration the fact that Caddy produced a denser turf than did PMAS and that disease development is usually more severe in dense turf, then Caddy may be more effective than the count of diseased spots indicates.

The alternating schedule of PMAS and Caddy was tested in the fall of 1959 in the hope of obtaining the more effective disease control features of the mercury and the dense-turf-producing qualities of the cadmium. The results were sufficiently encouraging early in the fall to justify recommending this schedule of treatments to golf course superintendents during the late winter of 1959-60. Although the commercial trials necessarily have been limited, they generally have given good results to date.

Miscellaneous Compounds

None of the miscellaneous materials tried in field tests gave sufficiently promising results to justify continued testing. They include Bordeaux, Bordeaux + Auragreen, and 1,2,-bis(ethyl sulfonyl)-1,2-dichloro ethylene. Thiram and Kromad were effective during mild attacks but not during severe ones. Mycostatin appeared somewhat promising at high rates but such rates were considered uneconomical, so further research with this material was discontinued. Several other materials were eliminated as a result of greenhouse testing.

CONCLUSIONS

Based on the preceding results, the current recommendations for control of Fusarium nivale on golf greens in western Washington are: 1) Regular treatment -- a bi-weekly alternating schedule of PMA (10% solution of phenyl mercury acetate at 3/4 ounce in 10 gallons water/1000 square feet) and cadmium chloride (20% solution at 1 ounce) during the spring and fall months with not longer than a monthly alternating schedule during summer and winter months; 2) Emergency treatment -- PMA (at above rates) at 1- to 2-week intervals until the fungus is brought under control. Adjustment of weekly, bi-weekly and monthly rates should be made according to anticipated or actual occurrence of Fusarium "weather" -- a knowledge soon acquired by superintendents in western Washington.

Nitrogen (as ammonium or calcium nitrate) can be added (at 1/8 pound nitrogen/1000 square feet) to the PMA during periods of stress (succulent grass and warm weather) to decrease phytotoxicity.

Higher rates of PMA and/or less dilution (5 gallons of water instead of 10) have been used with less danger and better control in drier areas, such as eastern Washington, where the grass is apparently less succulent.

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ROOT-KNOT NEMATODE ON KENAF IN GUATEMALA

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Kenaf (Hibiscus cannabinus) is becoming an important fiber crop in Guatemala. Parasitic nematodes constitute one of the problems related to the production of the fiber. In the Western Hemisphere, Meloidogyne incognita (Kofoid & White, 1919) Chitwood, 1949 was found attacking this fiber plant in Cuba (1). In Guatemala, Crandall[2] observed in 1959 and 1960 that nematodes constitute the limiting factor in the production of kenaf.

This report is based on a preliminary survey made in the Pacific coastal area of Guatemala. Soil and root samples collected at the kenaf program experimental plots of the Instituto Agropecuario Nacional, located at Escuintla, were studied. Soil samples were filtered in the laboratory using the Baermann funnel technique. Females were extracted directly from dissected kenaf roots. The technique described by Taylor, et al. (2) was used for the identification.

One species was identified in this preliminary survey, namely Meloidogyne incognita acrita Chitwood, 1949. This appears to be the first report of the species in Guatemala. Nematode attack is evident in the field by definite almost circular areas. Aerial symptoms produced by the root-knot nematode in the kenaf plant are stunting and foliar chlorosis. The destruction of secondary roots and the presence of galls of different sizes in the root system is evident in affected plants (Fig. 1). To solve this problem the use of special cultural practices and biological control are being considered.

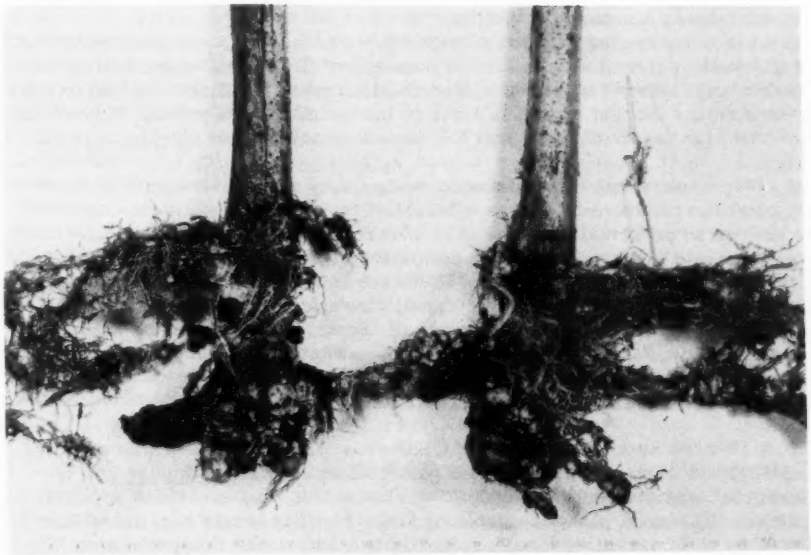

FIGURE 1. Symptoms of root-knot nematode attack in older roots of kenaf.

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[1]Acknowledgment is given to Bowen S. Crandall and Augusto Utrera for furnishing material from the kenaf experimental grounds, and Guillermo Lucero for the preparation of the photograph.
[2]Personal communication.

CONTROL OF PEPPER BACTERIAL SPOT BY FERTILIZER AND BY FOLIAR SPRAYS[1]

D. F. Crossan, D. J. Fieldhouse, P. P. Burbutis, W. W. Townsley, Jr., and Robert VanDenburgh[2]

Abstract

Three separate tests, one on a commercial basis, were carried out to determine the influence of fertility level and copper fungicides on the incidence and severity of bacterial spot of pepper caused by Xanthomonas vesicatoria. The use of from 1500 to 5000 pounds of 10-10-10 analysis or 3500 pounds of 6-12-12 analysis fertilizer markedly retarded leaf abscission and fruit spot of pepper due to infection by the bacterium. Leaf drop and fruit infection were severe where 500 to 1000 pounds of 10-10-10 or 1000 pounds of 6-12-12 fertilizer/acre was used. The use of foliar sprays of Tribasic Copper Sulfate or Bordeaux mixture gave excellent reduction in bacterial spot infection on plants growing at the latter levels of fertilizer.

INTRODUCTION

The production of peppers (Capsicum annuum) in Delaware has been seriously hampered in recent years. The combination of the European corn borer (Pyrausta nubilalis), the bacterial spot organism (Xanthomonas vesicatoria), and a series of summers with very heavy rains, has resulted in extensive reductions in yields.

Preliminary work at this station indicated that the corn borer was controlled by weekly applications of DDT (1) and bacterial spot was reduced by sprays of Tribasic Copper Sulfate (2). A recent report by Taylor and Dobson (4) indicated that the amount of fertilizer applied to pepper had a direct influence on the amount of leaf fall due to bacterial spot infection. Nayudu and Walker (3) have shown that high levels of N, P, and K reduced bacterial spot on tomato in controlled nutrient culture.

In the summer of 1960, several experiments were designed by the Departments of Plant Pathology, Entomology and Horticulture to compare levels of fertilization, with and without pesticide sprays, for control of bacterial spot and corn borer and the overall effect of fertility levels on yield. Three separate tests were carried out: one test on a commercial basis in cooperation with the H. P. Cannon Co. of Bridgeville, Delaware; one at the University Substation at Georgetown, Delaware; and one at the University Farm, Newark, Delaware. This report summarizes the data with respect to bacterial spot control. Summarizations of the data on insect control and plant response to fertilization will appear elsewhere.

METHODS AND MATERIALS

Bridgeville Test: A 10-acre section of a field of California Wonder peppers was selected for the test site. A split plot design was utilized with pesticide sprays superimposed on fertility levels. Each main plot was 400 feet in length, 70 feet in width, and contained approximately 2600 pepper plants. The main plots consisted of three fertility levels replicated four times: (a) 1000 pounds of 6-12-12 fertilizer/acre applied in two 500-pound increments on May 15 and June 8; (b) 2000 pounds of 6-12-12 fertilizer/acre applied in four 500-pound increments on May 15, June 8, June 30, and July 19; (c) 3500 pounds of 6-12-12 fertilizer/acre applied in one 500-pound increment on May 15 and three 1000-pound increments on June 9, June 30, and July 19. All of the fertilizer was applied as side-dressings. The pesticides were applied to half of each replicate plot of each of these fertility levels with a John Bean Air Blast machine which delivered 50 gallons of spray/acre. The plots were sprayed from two opposite sides with the following materials and concentrations/acre at the dates indicated: dichloro diphenyl trichloroethane (DDT)+ zinc ethylene bisdithiocarbamate (zineb) (Dithane Z-78) + Tribasic Copper Sulfate (4-2-4 pounds/acre) on July 15 and July 17; DDT+ 6, 7, 8, 9, 10, 10-hexachloro-1, 5, 5a, 6, 9, 9a, -hexahydro-6,9-methano-2, 4, 3-benzodioxathiepin-3-oxide (Thiodan) + Tribasic Copper

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Sulfate (4-1-4 pounds/acre) on July 29 and August 1; DDT + Thiodan + manganese ethylene bisdithiocarbamate (maneb) (Dithane M-22) + Tribasic Copper Sulfate (4-1-1 1/2-3 pounds/acre) on August 5 and August 11; DDT + maneb + Tribasic Copper Sulfate (4-1 1/2-3) on August 18, 25, and September 1, 1960.

Natural field infection was detected on July 29. Periodic ratings were made for defoliation due to bacterial leaf spot. The red ripe fruit were harvested on August 24, September 6, 23, and October 6; the green fruit were picked on October 10. The fruit were sorted into marketable, number two, and cull grades.

Georgetown Test: A split-plot design was used. The pepper variety was a Delaware selection called Calcom (California Wonder type) with resistance to tobacco mosaic virus. Five fertility levels were compared, using 10-10-10 fertilizer/acre as follows: (a) 1000 pounds with 500 pounds drilled in on May 19 and the remainder sidedressed on June 10 and July 8; (b) 1500 pounds, 500 pounds drilled in on May 19 and the remainder sidedressed on June 10, June 24, July 8 and July 22; (c) 2000 pounds, 500 pounds drilled in May 19 and the remainder sidedressed on June 10, June 24, July 1, July 8, July 22 and July 29; (d) 5000 pounds, 500 pounds drilled on May 19 and the remainder sidedressed on June 10, June 17, June 24, July 1, July 8, July 15, July 22, July 29, and August 5; (e) 1500 pounds, 500 pounds drilled on May 19 and the remainder sidedressed on June 10, July 8 and July 22 plus an additional 1750 pounds of 0-20-0 sidedressed on June 17.

Each plot consisted of four rows 40 feet in length with plants 18 inches apart in the row. Bordeaux mixture (4-4-100) was applied on July 11, 20, and 28 and Bordeaux (8-8-100) on August 5, 12, and 15 to half of the rows at each fertility level in each of the five replicates. All of the transplants were inoculated with the bacterial spot organism prior to transplanting. The disease became evident early in the season and was uniformly present in the plots prior to first spray applications of Bordeaux. Data were obtained on percentage defoliation, fruit infection, and yield of marketable fruit.

Newark Test: At this location four fertility levels were compared in a randomized, replicated block design. No copper-type sprays were applied. The fertilizer rates were as follows: (a) 500 pounds of 10-10-10 fertilizer/acre turned down with rye on May 4 and no further fertilizer added; (b) 1000 pounds 10-10-10/acre applied -- 500 pounds on May 4 and 250-pound increments on June 18 and Aug. 3; (c) 1500 pounds 10-10-10/acre applied in 500-pound/acre increments on May 4, June 18, and August 3; (d) 3000 pounds/acre 10-10-10 fertilizer applied in 500-pound increments on May 4, June 18, and 1000-pound increments on July 18, and August 3. In all cases, the May 4 application was turned under with a rye cover crop and all subsequent applications were made as side-dressings. Each plot consisted of six rows 30 feet in length, replicated four times. The plants were inoculated with the bacterial spot organism prior to transplanting. Data were obtained on percentage defoliation, fruit infection, and marketable yields.

RESULTS

Bridgeville Test: The data in Table 1 show the estimated defoliation at the various levels of fertility in both sprayed and unsprayed subplots and the number of green fruit infected with the bacterial spot organism in a five-basket subsample. It is apparent that the 2000-pound and 3500-pound rates of either the 10-10-10 or 6-12-12 fertilizer/acre markedly reduced the amount of leaf fall due to bacterial spot. The use of nine sprays containing Tribasic Copper Sulfate had the added effect of reducing defoliation to more than half of that due to fertility level alone. The decrease in the incidence, as well as severity, of leaf spot was in turn reflected in the number of fruit classified in a cull or a number 2 category. The addition of the copper sprays markedly reduced fruit infection at all fertility levels; the plants grown at high fertility had a significantly lower number of fruit infected compared with those at low fertility.

Georgetown Test: The data in Table 2 illustrated the marked influence that fertility, alone and in combination with foliar sprays, had on the number of leaves that abscised due to bacterial spot infection. Apparently the highest rate of fertilization markedly retarded abscission, with or without the use of foliar sprays. Further, the use of six sprays of Bordeaux mixture markedly reduced the leaf fall at the lower fertility levels. This is emphasized in Table 3 which indicates the relative percentage total leaf loss in the latter part of September due to bacterial spot.

Both the count of abscised leaves plus the estimation of percentage defoliation are substantiated by the count of the number of red and green marketable-sized fruit that were severely infected with Xanthomonas vesicatoria. Table 4 shows a significant decrease in cull fruit due to high fertility alone and a significant reduction in infection when Bordeaux was applied to plants

Table 1. The influence of fertility level and sprays on defoliation and fruit infection of pepper due to bacterial spot. Bridgeville, Delaware, 1960.

Fertility level (pounds N-P-K/acre)	% defoliation[a]		Av. no. inf. green fruit[b]	
	sprayed	unsprayed	sprayed	unsprayed
60-120-120	8	28	13.3	70.3
120-240-240	7	16	19.0	56.8
210-420-420	5	11	11.8	42.8

LSD (.05) for infected fruit, within sprayed or unsprayed, 11.3; between sprayed and unsprayed, 8.3

[a] Estimations made on 9/23/60 on four 50-foot sections of each 200-foot sub-plot replicate and averaged.

[b] Represents a five-basket sub-sample from each sub-plot replicate at the last harvest (green fruit) on 10/10/60.

Table 2. The influence of fertility and Bordeaux mixture on leaf abscission due to bacterial spot infection. Georgetown, Delaware, August 11, 1960.

Fertility level (pounds N-P-K/acre)	Average number abscised leaves[a]	
	sprayed	unsprayed
100-100-100	135	179
150-150-150	121	184
200-200-200	109	144
500-500-500	61	68
150-500-150	130	188

[a] Average number of leaves on the ground under each 2-row sub-plot of 40 plants in each of five replicates.

Table 3. The influence of fertility level and Bordeaux mixture upon defoliation of pepper due to bacterial spot. Georgetown, Delaware, September 25, 1960.

Fertility level (pounds N-P-K/acre)	Average % defoliation[a]	
	sprayed	unsprayed
100-100-100	9.5	21.0
150-150-150	8.1	18.0
200-200-200	9.0	14.8
500-500-500	3.8	5.3
150-500-150	8.8	21.0

[a] Estimated average total % leaf loss per 2-row sub-plot of 40 plants in each of five replicates.

Table 4. The influence of fertility level and Bordeaux mixture upon the total number of cull fruit due to bacterial spot. Georgetown, Delaware, 1960.

Fertility level (pounds N-P-K/acre)	Average no. cull fruit per replicate	
	sprayed	unsprayed
100-100-100	14.4	47.4
150-150-150	14.8	44.0
200-200-200	14.2	40.0
500-500-500	4.6	12.4
150-500-150	15.2	34.8

LSD (.05). Within treatments, 7.9; between sprayed and unsprayed, 7.8

at this 5000-pound/acre fertilization rate. It is interesting that the addition of a high level of phosphorus to a moderate N-K level significantly reduced severe fruit infection but not leaf fall in unsprayed rows. This effect should be investigated more thoroughly since the data to date do not adequately separate the individual effect of N, P, or K on infection. Spraying with Bordeaux significantly reduced fruit infection at all levels of fertility, although barely so at the highest fertilization rate.

Table 5. Estimation of the effect of fertilization levels on defoliation due to bacterial spot. Newark, Delaware, 1960.

Fertility level (pounds N-P-K/acre)	Average % defoliation[a]	
	August 3	August 22
50-50-50	27.2	35.9
100-100-100	21.9	24.0
150-150-150	5.0	6.6
300-300-300	1.0	2.3

[a] Value obtained by estimating the defoliation in each row of each 6-row plot in the four replicates and averaging the total value.

Table 6. The influence of fertility level on total number of pepper fruit classed as cull due to bacterial spot. Newark, Delaware, 1960.

Fertility level (pounds N-P-K/acre)	Number of cull fruit (average of four replicates)
50-50-50	66.5
100-100-100	48.0
150-150-150	32.5
300-300-300	8.3
LSD (.05)	31.7

Newark Test: The data obtained at this location, where no foliar sprays of copper fungicides were applied, are in agreement with those at other locations with respect to the influence of fertility level on defoliation and incidence of fruit infection. The data on the relative amount of defoliation are presented in Table 5. The 1000-pound/acre rate of 10-10-10 fertilizer did reduce the leaf drop but the effect was most evident at the 1500- and 3000-pounds/acre rates. This reduction in total infection and leaf fall is reflected in the total number of fruit harvested and classed as a cull due to bacterial spot infection. The constant and significant reduction in severe fruit infection as the amount of fertilizer/acre is increased is illustrated by the data in Table 6.

DISCUSSION

The data obtained at the three different locations indicate strongly that the use of a complete fertilizer to give about 120 to 150 pounds of nitrogen, phosphorus, and potassium/acre will materially reduce defoliation due to bacterial spot. In a summer of high light intensity, this should be reflected in less cull fruit due to sunscald. The addition of sprays of Tribasic Copper Sulfate or Bordeaux mixture at this level of fertilization may be expected to give a significant further reduction in leaf fall and fruit infection.

The specific influence of fertilization levels on total yield are not reported herein. However, at two locations (Newark and Georgetown) yields increased with increases in amount of fertilizer up to the 1500- and 2000-pounds of fertilizer/acre rate. Increases beyond these levels were not significant. At Bridgeville, the 2000- and 3500-pound rates reduced yield. This particular location went through a drouth during the period of most of the side-dress applications, whereas the other two locations did not.

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DELAWARE AGRICULTURAL EXPERIMENT STATION, NEWARK

CASTORBEAN DISEASES IN TEXAS IN 1960

Raymond D. Brigham[1]

Two diseases caused serious damage to castorbeans grown under irrigation in the High Plains area around Plainview, Texas in 1960. About 20,000 acres were planted to Baker 296, an early, dwarf-internode, inbred line. A seedling disease, not previously reported, and Alternaria leaf spot were the diseases of primary importance.

An outbreak of seedling disease occurred in the last 2 weeks of May shortly after the seedlings had emerged in the early-planted fields. Topsoil moisture was lacking in most fields. Air temperatures were rising daily under clear skies.

Aboveground symptoms were most evident on the cotyledonary leaves. The tip of one or both cotyledons became yellowed and chlorotic, and this chlorosis progressed toward the petiole. Diseased plants were usually stunted, and cotyledons were often curled or twisted. In advanced stages, one or both of the cotyledons became entirely chlorotic, the cotyledons and stem lost turgidity, and all aboveground parts appeared dying or dead.

Root systems of diseased plants were very abnormal, and appeared to be diseased. In most cases only a few primary roots appeared functional. In extreme cases, no attached functional roots could be recovered when seedlings were carefully dug (Fig. 1). These apparently rootless plants had lost or were in the process of losing cell turgidity.

FIGURE 1. Seedlings of Baker 296 castorbeans with damaged root systems and swollen, cracked hypocotyls. The first plant on the left in each row is normal; all others are diseased. Note the almost complete lack of roots on some seedlings.

Directly above the region of root attachment, the hypocotyl of most diseased plants was usually swollen 1 1/2 to 2 diameters larger than normal size. A longitudinal growth crack from 1/2 to 1 1/2 inches long was normally present in these swollen hypocotyls, and appeared to penetrate the cortex and stele. When only one cotyledon was visibly chlorotic, it was usually supplied by conductive tissues on the side of the hypocotyl where the crack occurred.

Attempts were made to isolate pathogens from the diseased plants. Dr. C. A. Thomas, Beltsville, Maryland obtained isolates of Rhizoctonia, but none was cultured that proved pathogenic in greenhouse inoculations on vigorous seedlings of Baker 296. An unidentified fungus, tentatively classed in the Fungi Imperfecti, was also consistently found in the diseased seedlings at College Station.

Rains fell in the castorbean-growing area the first week in June, and the majority of the diseased plants made a rapid recovery. A few fields were irrigated down the drill row before rains occurred, as the plants had not had a first cultivation to open a water furrow. Approximately 10,000 acres of early-planted castorbeans were affected by the seedling disease, but none was abandoned. Had timely rains not occurred, however, several hundred acres would likely have been lost.

A review of weather conditions from time of planting until recovery from the disease indicated the probable factors responsible for the seedling disorder. Soil temperatures warmed up sufficiently in late April for planting of castorbeans, but a succession of cool fronts with

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precipitation then lowered air and soil temperatures during the first 2 weeks of May. In the last half of May air and soil temperatures warmed quickly, and winds dried out the topsoil rapidly. No effective precipitation occurred during the latter part of May.

Most of the early root development of seedling castorbeans normally occurs shortly before and after emergence, but cold, wet soils at this time greatly reduced root development, and possibly allowed fungi to damage the root system. The cracking of the hypocotyl, which undoubtedly provided an entrance for soil organisms, likely occurred while the root system was still functioning, an oversupply of soil moisture was present, and low air temperatures slowed transpiration. When air and soil temperatures increased rapidly in late May, the damaged root system, much of which was then in dry soil, was inadequate to supply the moisture needed to maintain the plant. When rains came in early June after soil temperatures were up, most plants were then able to produce more roots and recover from the disorder. Late planted castorbeans were not damaged by this seedling disease, as they emerged after the cold, wet weather occurred.

Although isolates from the diseased seedlings were not pathogenic on vigorous plants of Baker 296 under greenhouse conditions, Rhizoctonia or other fungi may have been pathogenic on the weakened seedlings under field conditions. Probably these fungi were able to invade the cracks in the hypocotyl as well as to attack the roots. Isolates might be proven pathogenic if conditions could be duplicated as they occurred in the field.

Alternaria leaf spot, caused by Alternaria ricini (Yoshii) Hansford, caused serious losses in yield in fields of Baker 296 in 1960. Although it was not possible to correlate susceptibility of individual plants, it was noted that defoliation by Alternaria leaf spot was more serious in fields where the plants had weakened root systems partly due to the seedling disorder. Plants of Baker 296 in most fields were sufficiently defoliated and dried for mechanical harvest by October 15, whereas plants of Dawn and RA 348, an experimental line, were still green and in full foliage.

Losses in yield due to Alternaria leaf spot were estimated to be 25 to 35% in the Baker 296 fields that were harvested in October 1960. The disease also lowered yields an estimated 10 to 40% in Baker 296 fields in 1959, and 5 to 20% in 1958.

Susceptibility of castorbeans to Alternaria leaf spot appears to be partially correlated with earliness and an accompanying weak root system. Baker 296, a susceptible, early-flowering variety, develops a weak root system compared with resistant, late-flowering varieties. This is apparently due to competition for available photosynthate and/or other nutrients between the roots and developing capsules, after fruit set is initiated. When a plant sets more fruit than a weak root system can supply with nutrients, and unfavorable weather conditions occur, the plant undergoes stress and is predisposed to infection by Alternaria. Later planting of Baker 296 to allow less fruit set may avoid these predisposing factors.

Alternaria capsule mold, caused by the same fungus as above, was also prevalent in the High Plains area of Texas[2]. Above average rainfall with accompanying high relative humidity made conditions optimum for all types of capsule molds.

Bacterial leaf spot, caused by Xanthomonas ricinicola (Elliott) Dowson, was present on scattered plants early in the growing season, but did not spread from the initial infection.

Cercospora leaf spot, caused by Cercospora ricinella Sacc. & Berl., was observed on scattered plants in experimental plantings and in a few commercial fields of Baker 296.

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[2]Brigham, Raymond D., and Ben R. Spears. 1960. Castorbeans in Texas. Texas Agr. Exp. Sta. Bull. No. 954.

FREQUENCY OF COTTON PLANTS RESISTANT TO FUSARIUM WILT IN SOME LINES OF COTTON RESISTANT OR SUSCEPTIBLE TO BACTERIAL BLIGHT

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Abstract

Seven of nine lines of cotton developed for resistance to bacterial blight were found to contain much higher percentages of plants resistant to Fusarium wilt than did three lines susceptible to blight. The relationship of the resistant factors for each of these diseases has not been determined, but it would appear profitable to screen existing blight-resistant lines for resistance to Fusarium wilt and root-knot nematodes.

During the 1960 season a planting of bacterial-blight-resistant and blight-susceptible lines of two species of cotton, Gossypium hirsutum and G. barbadense, was grown inadvertently in an area of a field near Hollis, Oklahoma that was heavily infected with the Fusarium wilt fungus (F. oxysporum f. vasinfectum (Atk.) Snyd. & Hans.) and with the root-knot nematode (Meloidogyne incognita acrita Chitwood).

The reaction to Fusarium wilt of 10 bacterial-blight-resistant lines and also of 4 blight-susceptible lines is shown in Table 1. The criteria upon which the Fusarium-wilt grades are based are included as a footnote to Table 1. In order to classify a plant, the stem was cut diagonally to the center just above the soil line. Plants which showed no discoloration in the initial cut usually were found to contain a few very slightly discolored flecks in the xylem tissues of the root or other portions of the stem when examined more closely.

Table 1. Fusarium-wilt reaction of lines of cotton resistant or susceptible to bacterial blight.

Variety or designation	Bacterial blight reaction	Major gene conditioning resistance to blight	Reaction to Fusarium wilt: Number of plants in grade:[a] 1	2	3	4	5	Mean grade
1-10B	resistant	B_{I_n}	0	0	1	0	128	4.98
4-11	do.	B_L[b]	28	23	19	29	47	3.30
8-3	do.	b_7	25	29	17	18	93	3.69
1517BR-1	do.	b_7	3	18	12	10	5	2.92
Rex	do.	b_7	4	12	9	4	5	2.82
20-3	do.	B_N	27	53	32	16	54	3.09
(20-3 X 4Ac44)F_4	do.	B_N	0	3	1	3	8	4.07
Gregg	do.	?	0	0	0	0	21	5.00
Knight's G. barbadense	do.	B_1	10	46	25	2	4	2.36
Knight's G. barbadense	do.	B_2	28	59	22	9	7	2.26
Auburn 56	susceptible		68	44	28	8	4	1.92
Stoneville 62	do.		0	0	13	5	78	4.68
Acala 44	do.		1	7	19	27	117	4.47
Lankart 5[c]	do.		0	3	8	12	240	4.86

[a]Grade 1, plants with no apparent discoloration to a few minor flecks in the xylem tissues; Grade 2, flecks to slight streaks in the xylem; Grade 3, definite brown streaks in xylem, plants usually less fruitful; Grade 4, plants usually stunted, cambial region brown, dark continuous streaks in xylem; Grade 5, plants dead.

[b]Present data are not conclusive as to the genotype of 4-11, but a single dominant gene appears to be responsible for resistance to bacterial blight.

[c]Lankart 57 was not included with the lines of cotton described here, but was grown in a field in another area that was similarly affected.

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The Fusarium-wilt-grade distributions for Stoneville 62, Acala 44 and Lankart 57 are comparable to those of blight- and wilt-susceptible varieties that have been grown in past years. Usually a very few plants of such varieties appear to be resistant. Promising selections, nevertheless, have been derived from the variety Cluster which was of this type.

Auburn 56, a blight-susceptible variety developed by Smith and Tisdale (1) in Alabama for its resistance to Fusarium wilt and root-knot nematodes, usually shows excellent resistance to Fusarium wilt in Oklahoma. The percentage of susceptible plants in 1960 was somewhat higher than in past years and may have been due to impure seed.

With the exception of the variety Rex, none of the blight-resistant lines had been purposely selected for resistance to Fusarium wilt. Rex was bred by Moosberg (3) in Arkansas as a backcross involving a blight-resistant line of Stoneville 2B and Fusarium-wilt-resistant Empire as the recurrent parent.

The blight-resistant line 1-10B and the variety Gregg appear to be comparable in wilt susceptibility to the blight-susceptible varieties previously discussed. Whereas, the blight-resistant lines 4-11, 8-3, 1517BR-1, 20-3, (20-3 X 4Ac44)F_4, Knight's B_1, and Knight's B_2 contained many more Fusarium-wilt-resistant plants than would be expected of wilt-susceptible varieties.

Readings for resistance or tolerance to root-knot nematodes were not obtained for individual plants of all the different lines. However, plants that were examined usually showed much less injury from root-knot nematodes than did wilt-susceptible plants. Smith and Dick (2) have shown that resistance to Fusarium wilt in Upland cotton is partially conditioned by resistance to root-knot nematodes.

Whether there is an association between certain genes conditioning resistance to bacterial blight and resistance to Fusarium wilt is yet to be determined. It would appear, however, that it might be profitable to screen blight-resistant breeding material for resistance to Fusarium wilt and root-knot nematodes.

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THE EFFECT OF VARIOUS DICHLONE TREATMENTS ON THE GROWTH, YIELD, AND DISEASE INCIDENCE OF POTATOES AND TOMATOES IN NEW HAMPSHIRE[1, 2]

James D. Bilbruck and Avery E. Rich[3]

Abstract

The yields of potatoes and tomatoes were reduced when plants were sprayed with high concentrations of dichlone (1 pound or more of Phygon XL per 100 gallons of water). However, concentrations of 1/4 or 1/2 pound Phygon XL (50% dichlone) did not significantly reduce yields of potatoes or tomatoes. The 1/4-pound rate controlled early blight of potato and tomato and late blight of potato fairly well. Midday applications of dichlone were more injurious than dichlone sprays applied in the morning or evening. Dichlone sprays did not increase the top weight of potato plants. These results indicate that potato yield reductions are not due simply to a reduction in either the synthesis or translocation of carbohydrates.

INTRODUCTION

Dichlone (2,3-dichloro-1,4-naphthoquinone) is one of the oldest and most effective organic fungicides on the market. It is commonly marketed as Phygon XL, which contains 50% dichlone. Probably it would have found wider use if it were not phytotoxic under certain conditions.

Dichlone is effective for the control of late blight (Phytophthora infestans) of potato and tomato and for the control of early blight (Alternaria solani), tomato septoriosis (Septoria lycopersici), and anthracnose (Colletotrichum phomoides) of tomato. However, under certain conditions it reduces the yield of potatoes and produces superficial blackening of tomato fruits.

This work was undertaken to obtain more definite information concerning the effect of dichlone on potatoes and tomatoes under New Hampshire conditions.

REVIEW OF LITERATURE

Several workers have reported success in the use of dichlone for the control of several diseases of potatoes. However, because of changes in formulation by the manufacturers and incomplete data in some of the reports on the use of Phygon, it is frequently impossible to determine the exact amount of active ingredient used.

Ruehle (13, 14) states that Phygon XL, when used at 0.75 pound per hundred gallons of water "is a very promising fungicide for preventing potato seed-piece decay (Fusarium spp.); however, when Phygon was used for control of potato late blight (at 1-100), it was found to be very effective in controlling the disease, but it was toxic to the plants and the yields were low." Olmsted (11) states that, when used at the rate of 0.5% as a treatment for seed-piece decay of potato, "Phygon-----increased yields appreciably." Richards (12) found no correlation between yield and disease control in a comparison of 10 fungicides, one of them being Phygon used at the rate of 2-100. Data presented by Hoyman (9) indicate that Phygon used at the recommended rate did not appreciably depress the yield of Bliss Triumph potatoes in the absence of early and late blight. Davidson and Rich (6, 7) were among the first to show that Phygon depressed potato yields. They found that Phygon was effective in controlling late blight in Rhode Island in 1945 (2-100) and 1946 (1/2-100), but that it depressed the yield both years. Callbeck (4) found that Phygon XL (1-40) delayed maturity of Green Mountain potatoes in Canada in 1948. Bonde and Snyder (3) found that Phygon (1-100) reduced early blight leaf injury from 15.1 to 7.3% and to 3.4% when dichlorodiphenyl trichloroethane (DDT) was added; they also state that the Phygon plots were actively growing on September 20, when the Bordeaux-treated plants were dwarfed and the lower leaves dead.

Harrison (8) found that Phygon (1-100) is effective for the control of late blight in tomato seedling beds in Florida. Phygon was also found effective against the same disease in Connecticut (2). Andrade (1) states that Phygon is effective for the control of tomato septoriosis, A. solani, and P. infestans. But he also states, "It is, however, apt to cause severe damage to

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[2] Based on a portion of a thesis submitted by the senior author to the Graduate School, University of New Hampshire, in partial fulfillment of the requirements for the degree of Master of Science.

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certain other plants, for example, vine and fig, and should be used with great care; the prescribed concentration of 120 g in 100 liters can safely be reduced to 100 g or even to 60 g with a good spreader and adhesive, without loss of efficiency." Phygon XL (3/4-100) has been used in Florida (10) for the control of tomato late blight, as an alternate spray with disodium ethylene bisdithiocarbamate (nabam) plus zinc sulfate, to cut down injury caused by the latter fungicide. In a recent report, Darby (5) states that dichlone (3/4-100) "was the most effective material tested for the control of both gray mold (Botrytis cinerea) and ghost spot; however, it was ineffective against gray leaf spot (Stemphylium solani)." Schroeder (15, 16) states that Phygon (86% active, at 1-100 and 2-100) gave good control of early and late blights and of anthracnose of tomato, but left a superficial blackening on the fruit. This blackening was restricted to the epidermis and was worse at the higher concentration of Phygon.

FIELD EXPERIMENTS

Potatoes and Tomatoes, 1952

Procedure: New Hampshire Victor tomato seedlings were set out in the field during the first week of June. The plot arrangement was a 10 x 10 latin square, with a single plant serving as a plot. The rows were 4 feet apart, with the plants 3 feet apart in the row.

One hundred Green Mountain potatoes were planted near the tomatoes in a randomized block arrangement, 4 feet apart each way; the blocks were five plants wide, with 10 plants in each block.

The potatoes and the tomatoes received the same treatments, as indicated in Tables 1 and 2. Treatments were designed to test the effects of different concentrations of dichlone, of spraying at different daily intervals, and of spraying at different times of the day. Early morning and evening sprays were included to test whether dichlone interfered either with the production or the translocation of carbohydrates.

The sprays were applied with a Brown 4-gallon hand sprayer. Potatoes and tomatoes received like treatments and were sprayed at the same time with enough spray to cover the plants. Copper oxychloride sulfate (COCS) was used as a standard treatment for comparison.

Fungicides were applied from July 8 until September 5, at the indicated intervals. Twenty sprays were applied at 3-day intervals; 11 sprays were applied at 6-day intervals; and 6 sprays were applied at 12-day intervals.

DDT dust or spray was used to control flea beetles, tarnished plant bugs, aphids, and leafhoppers, as found necessary.

Results: The tomatoes were harvested periodically as they ripened. The 1952 tomato yield data could not be subjected to analysis of variance, since approximately 20% of the plants produced parthenocarpic fruit on extremely vigorous, over-sized plants. Four of these so-called "bull plants" were found in treatment 2 alone. Only treatments 4 and 6 did not contain any "bull plants." The 1952 average yield per tomato plant for the normal plants in each treatment is shown in Table 1.

Table 1. Effect on yield of spraying tomato plants with Phygon XL at different concentrations, at different daily intervals, and at different times of day, compared with effect on yield of COCS and no treatment, 1952.

Treatment	Rate (pounds/100 gallons)	Time interval (days)	Average yield (kg/plant)
1. Phygon XL	1/8	6	6.2
2. do.	1/4	3	6.6
3. do.	1/2	6	6.6
4. do.	1	6	5.3
5. do.	1/4	6 (noon)	6.6
6. do.	1/4	12	7.8
7. do.	1/4	6 (6 a.m.)	6.9
8. do.	1/4	6 (7:30 p.m.)	7.3
9. Check	no treatment		6.4
10. COCS	3	6	6.8

The tomato data for 1952 show a trend which indicates that the greater the concentration of dichlone, the lower the yield. Treatments 2 and 3 received the same amount of dichlone, but

treatment 2 received half the concentration, with twice as many applications; the average yield for these two treatments was the same. The highest concentration of dichlone produced the lowest yield.

The yield data for the potato plots in 1952 (Table 2) have been subjected to analysis of variance. The plants receiving the highest concentration of Phygon produced the lowest yield. None of the other treatments, except COCS, significantly reduced yields below those of the check plants. Top weights, both fresh and dried, were recorded for the 1952 potato plots (Table 2). None of the treatments differed significantly from the check plants in either green or dry top weights. The plants sprayed with COCS were the lightest in weight.

Table 2. Effect on yield and top weights of spraying potato plants with Phygon XL at different concentrations, at different daily intervals, and at different times of day, compared with effect on yield of COCS and no treatment, 1952.

	Treatment	Rate (pounds/100 gallons)	Time interval (days)	Average yield (kg/plant)	Average top weight (kg/plant) fresh	dry
1.	Phygon XL	1/8	6	2.93	3.05	0.50
2.	do.	1/4	3	2.82	3.34	0.55
3.	do.	1/2	6	2.63	2.88	0.42
4.	do.	1	6	1.80**	2.87	0.40
5.	do.	1/4	6 (noon)	2.66	2.79	0.40
6.	do.	1/4	12	2.82	2.81	0.44
7.	do.	1/4	6 (6 a.m.)	2.87	2.90	0.44
8.	do.	1/4	6 (7:30 p.m.)	3.04	3.24	0.49
9.	Check	no treatment		3.01	2.94	0.46
10.	COCS	3	6	2.24*	2.27*	0.34*
*	Diff. necessary for significance (19:1)			0.72	0.52	0.19
**	Diff. necessary for significance (99:1)			0.95		

Potatoes and Tomatoes, 1953

Procedure: The experiment with potatoes and tomatoes was repeated in 1953, using a 9 x 9 latin square for each crop. Since there were so many tomato plants which produced parthenocarpic fruits among the New Hampshire Victor tomatoes used in 1952, the Marglobe variety was chosen for 1953. Green Mountain potatoes were again used. The concentrations of dichlone in the sprays were increased in an attempt to obtain greater differences between treatments.

The sprays were applied with a Brown 4-gallon sprayer, as in 1952, beginning July 10 and ending September 14. Twenty-two applications were made at 3-day intervals; 12 applications were made at 6-day intervals; and 6 applications were made at 12-day intervals. DDT dust or spray was again used, as needed, to control insects.

Results: Results are presented in Table 3. Tomato plants receiving the lowest concentration of dichlone produced the largest yields; plants sprayed with COCS produced the lowest yields.

The lowest potato yields were produced by plants receiving the highest concentration of dichlone; plants receiving the lowest concentration of dichlone produced one of the highest yields.

Top weights of the potatoes were not taken in 1953 because the tops were killed by frost 2 weeks before harvest.

Late blight in the tomato plots was not a factor either year. No late blight infection appeared in the 1952 potato plots, but in 1953 the unsprayed check plants averaged 7% defoliation - a low infection for unsprayed check plants.

EFFECT OF PHYGON ON GREENHOUSE-GROWN POTATO PLANTS

Procedure: On January 27, 1954 twelve Green Mountain potatoes were cut in halves, making 24 seed pieces which were dipped in Phygon XL (1/2 pound per 100 gallons) to protect against rot organisms, and placed in moist peat moss to suberize.

On February 3, 1954 20 of the potato seed-pieces were planted individually in pails of soil obtained from the 1953 potato and tomato field plots. No fertilizer was added at the time of

Table 3. Effect on yield of spraying Marglobe tomato and Green Mountain potato plants with Phygon XL at different concentrations, at different daily intervals, and at different times of day, compared with effect on yield of COCS and no treatment, 1953.

	Treatment	Rate (pounds/100 gallons)	Time interval (days)	Average yield (kg/plant) potatoes*	tomatoes**
1.	Phygon XL	1	6 (6 a.m.)	3.58	12.48
2.	do.	1	3	3.08	10.62
3.	do.	1	12	3.00	11.44
4.	do.	1/2	6	3.64	12.73
5.	do.	2	6	2.64	10.66
6.	do.	1	6 (noon)	2.86	10.48
7.	do.	1	6 (7 p.m.)	3.72	11.33
8.	COCS (Std.)	3	6	3.22	8.85
9.	Check	no treatment		3.40	10.42

* Diff. necessary for significance at 19:1, 1.63 kg.
** Diff. necessary for significance at 99:1, 2.22 kg.

planting.

Beginning on February 26, 10 of the potatoes were sprayed with Phygon XL (1 pound per 100 gallons) at approximately weekly intervals until April 8, a total of seven sprays. All of the potatoes were fertilized with ammonium nitrate, at the rate of 2 teaspoonfuls per gallon of water; this was done three times (February 26, March 8, and March 22).

The potato tops were cut April 29, fresh weights were taken, and the tissue was then dried in an oven at 75° C for 3 days. The tubers were harvested on May 3.

Results: Well-fertilized potato plants apparently were not injured by dichlone, even though applied at twice the recommended concentration. The average differences (Table 4), did not vary greatly; therefore, in this experiment dichlone apparently did not have a significant effect either on tuber yield or on top growth.

Table 4. Average weight of tops and tubers of 10 greenhouse-grown potato plants which received seven sprays of Phygon XL (1 pound per 100 gallons) compared with 10 untreated check plants.

Treatment	Top weight (kg) fresh	dried	Tuber weight (kg)
Phygon XL	0.211	0.022	0.246
Check	0.198	0.021	0.236

EFFECT OF DICHLONE AND OTHER FUNGICIDES ON POTATO AND TOMATO DEFOLIATION AND YIELDS

Procedure: Evaluation tests of several fungicides were conducted during the summers of 1952 and 1953. Phygon XL was compared with several other fungicides for the control of early blight and late blight of potato and tomato.

Results: Phygon XL gave satisfactory control of early blight in 1952; late blight was not a factor in 1952. Control of both early and late blight was satisfactory in 1953, without significant reduction in yield.

A comparison of percentages of defoliation and yields of plants treated with Phygon XL manganese ethylene bisdithiocarbamate (maneb), nabam plus zinc sulfate, N-trichloromethylmercapto-4-cyclohexene-1,2-dicarboximide (captan), COCS, and of the untreated check plants is given in Table 5.

DISCUSSION AND CONCLUSIONS

The results of these experiments show that yield of potatoes was reduced when they were sprayed with Phygon XL at the rate of 1 or 2 pounds per 100 gallons of water. This substanti-

Table 5. Effect of various fungicides on the defoliation and yields of Green Mountain potatoes and New Hampshire Victor tomatoes in 1952 and 1953.

Treatment	Rate (pounds/100 gallons)	Potatoes				Tomatoes			
		% defoliation[a]		yield/plot[b] (kg)		% defoliation[c]		yield/plot[d] (kg)	
		1952	1953	1952	1953	1952	1953	1952	1953
Phygon XL	1/4	6	15	2.2	5.0	14	12	6.3	9.1
Phygon in alcohol	1/8	13		2.3		15		6.8	
Maneb	2	4	4	2.1	5.6	15	11	5.3	11.8
Nabam + $ZnSO_4$	2 qt +1 lb	5	9	1.7	5.3	17	12	4.7	12.0
Captan	2	5	21	2.3	4.5	6	10	4.6	13.7
COCS	3	15	24	2.0	4.0	19	21	4.3	11.5
Check		15	94	1.8	2.1	35	34	5.3	11.9
Standard error of mean				0.3	0.5				2.7

[a]Average estimated defoliation due to Alternaria solani in 1952 and Phytophthora infestans in 1953.
[b]Average yield of 6 single-plant plots in 1952 and 4 four-plant plots in 1953.
[c]Average estimated defoliation due mostly to Alternaria solani both years.
[d]Average yield of 6 single-plant plots in 1952 and 4 two-plant plots in 1953.

ates the earlier work of Davidson and Rich (6, 7) and others. Tomato yields were also depressed somewhat when plants were sprayed at these high concentrations. However, when applied at 1/4 or 1/2 pound per 100 gallons, Phygon XL did not significantly reduce yields of potatoes or tomatoes. The 1/4-pound rate gave fairly good control of early blight of potato and tomato and late blight of potato under conditions which were moderately favorable for disease development. The 1/8-pound rate dissolved in alcohol (Table 5, treatment 2) was unsatisfactory.

It was assumed that if sprays applied in the evening should reduce yields, this would indicate that dichlone interferes with the translocation of carbohydrates; and if morning applications should reduce yields, the reduction might be due to interference with the synthesis of carbohydrates. As the plots receiving these treatments yielded better than those receiving midday applications, apparently plants are more subject to injury in the middle of the day, and yield reductions cannot be explained simply as a reduction in either the synthesis or translocation of carbohydrates. The fact that dichlone sprays did not increase the top weight of potato plants is further evidence that the reduction of tuber weight is not the result of interference with the translocation of carbohydrates from the leaves to the tubers.

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SURVIVAL IN THE PLANTATION OF EASTERN REDCEDAR SEEDLINGS INFECTED WITH PHOMOPSIS BLIGHT IN THE NURSERY

C. S. Hodges and H. J. Green[1]

Abstract

Nursery seedlings of eastern redcedar infected with Phomopsis juniperovora were planted in the field to correlate field survival with degree of infection at planting time. Seedlings with as little as 10% infection had a survival rate of only 24 to 30% at the end of two growing seasons. The greatest loss was due to reinfection of previously diseased seedlings. Little spread of the disease was observed between diseased and healthy seedlings in the plantation. Viable conidia of the causal organism were recovered from branches that had been dead for as long as 18 to 24 months, indicating a potential for long survival of the fungus in infected material.

Phomopsis blight, caused by Phomopsis juniperovora Hahn, is a limiting factor in the production of eastern redcedar (Juniperus virginiana) seedlings throughout the eastern and midwestern United States. The disease often results in high seedling mortality in the nursery beds, and in years of high disease incidence 50% or more of the surviving seedlings may have some degree of infection. Many nurseries in the past have shipped diseased seedlings, especially those with light infection, although no information was available as to continued development of the disease in the plantation. The purpose of this study was to determine the survival and further disease development in a plantation of seedlings that had been initially affected with Phomopsis blight in the nursery.

PROCEDURE

The seedlings used in these studies were obtained from the Clayton Nursery near Clayton, North Carolina. Four classes of seedlings were selected, based on degree of infection with P. juniperovora: 1) apparently healthy seedlings, 2) seedlings with less than 10% of the top dead, 3) seedlings with 10 to 50% of the top dead, and 4) seedlings with over 50% of the top dead. The seedlings were planted on the Bladen Lakes State Forest near Elizabethtown, North Carolina in January 1958. Four replications of 25 seedlings each were used for each class, with the seedlings spaced 4 feet apart.

In January 1959 a similar planting was established adjacent to the first. For this planting, five disease severity classes were used, with the 10 to 50% infection class used in the first planting divided into two classes having 10 to 25 and 26 to 50% infection, respectively. Four replications of 25 seedlings each were used for each class. Data on survival and disease development were taken periodically in both plantings.

RESULTS AND DISCUSSION

Disease severity of nursery seedlings was correlated with subsequent survival in the plantation (Tables 1 and 2). In the 1958 planting heavy mortality occurred in the 10 to 50 and over 50% infection classes the first year in the plantation, whereas in the 1 to 9% class survival was only slightly lower than in the healthy seedlings. By June of the second growing season, however, considerable mortality had occurred in all classes with the exception of the healthy seedlings. This same relationship also held true for the 1959 planting, although survival in the healthy seedling class was lower than in the 1958 planting.

The disease progressed rapidly during the first growing season in all of the seedlings that were infected at time of planting. In most instances this was due to new infections by conidiospores taking place on lower branches, although some growth of the fungus down branches and stems took place. This downward growth usually stopped when a secondary branch was reached.

[1]Plant Pathologist, Southeastern Forest Experiment Station, Forest Service, United States Department of Agriculture, in cooperation with the North Carolina Agricultural Experiment Station; and Pest Control Forester, North Carolina Division of Forestry, respectively. Appreciation is expressed to Mr. Graham Chamblee and other employees of the Bladen Lakes State Forest for their help in establishing the plantings.

Table 1. Survival of redcedar seedlings planted in January 1958 having different degrees of infection by P. juniperovora.

Disease severity at planting	% survival				
	Jan. 1959	June 1959	Oct. 1959	Mar. 1960	Aug. 1960
Healthy	99	87	87	84	83
1-9% of top dead	89	25	24	23	23
10-50% of top dead	71	11	8	6	6
50+% of top dead	55	3	3	3	3

Table 2. Survival of redcedar seedlings planted in January 1959 having different degrees of infection by P. juniperovora.

Disease severity at planting	% survival			% surviving seedlings free of new infections[a]
	Oct. 1959	Mar. 1960	Aug. 1960	
Healthy	95	81	72	82.9
1-9% of top dead	82	55	30	18.2
10-25% of top dead	75	41	17	9.8
26-50% of top dead	60	21	8	9.5
50+% of top dead	27	15	8	0.0

[a] March 1960.

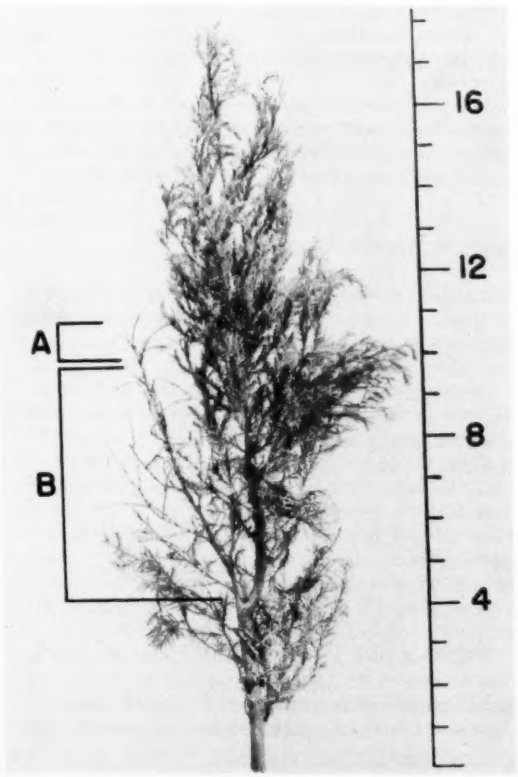

FIGURE 1. Seedlings of eastern redcedar showing recovery from Phomopsis blight. A -- Portion of seedling dead at time of planting. B -- Portion of seedling which died during first year. (Scale in inches)

If this occurred on the main stem, the branch below the infection usually turned upward and became the new leader (Fig. 1). Most of the surviving plants from the groups infected at the time of planting show the condition illustrated. The majority of these plants appeared to have recovered from the disease, especially those in the 1958 planting. Their height growth, however, was only about half that of the healthy plants.

Most of the seedlings that died during the second growing season were severely diseased at the end of the first growing season. It was not unusual to find plants with 20 or more separate infections and in some instances with only a few small living branchlets.

During both 1959 and 1960, new infections were observed as early as February. A check of the native redcedar in the surrounding area indicated no evidence of blight; thus, all the new infections occurring in the plantation presumably originated from infected trees within the plantation. Most of the new infections occurred on previously diseased plants (Table 2) and the incidence of spread from infected to healthy plants was low. This was to be expected since the fungus is considered to spread locally mainly by splashing rain. Viable conidia of the fungus were recovered from pycnidia in twigs that had been dead for as long as 18 to 24 months, indicating that the fungus is capable of overwintering in infected tissue.

The results of the study indicated that all infected seedlings in the nursery should be culled from planting stock, since the chance of their surviving, or making an acceptable tree, is small. Although there was no evidence that spread of the disease from infected to healthy seedlings in the plantation occurs to any great extent, limited spread does occur and could result in additional loss.

One factor that was not considered during the study was the possible spread of the disease during transit or storage. Watering bundled seedlings in storage or transit creates favorable conditions for sporulation and dispersal of the fungus, and if the temperature is favorable for the development of the disease, heavy losses may occur. This factor may be more important from the standpoint of spread of the disease from infected to healthy seedlings than that which occurs in the plantation.

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A SURVEY OF CUCURBIT VIRUSES IN THE LOWER RIO GRANDE VALLEY OF TEXAS: PRELIMINARY REPORT

D. M. McLean and H. M. Meyer[1]

Summary

The viruses affecting cucumber, squash, cantaloupe, Honey Dew, and watermelon in the Lower Rio Grande Valley of Texas were indexed on selected test plants during two spring and one fall seasons of 1959-60. From 140 virus samples assayed, 124 contained tobacco ringspot virus (TRSV), 18 watermelon mosaic virus (WMV), 10 squash mosaic virus (SqMV), and 2 cucumber mosaic virus (CMV). TRSV was isolated frequently alone or in combination with the other viruses. The prevalence of TRSV and the rare occurrence of other cucurbit viruses indicate that the virus complex in the Lower Rio Grande Valley of Texas is very different from the cucurbit virus complexes reported elsewhere. Such differences in prevalence of the several cucurbit viruses may be associated with differences in relative abundance of vectors, reservoir hosts, both, or other unknown factors.

Widely distributed cucurbit viruses within the United States include cucumber mosaic virus (CMV), watermelon mosaic virus (WMV), squash mosaic virus (SqMV), and tobacco ringspot virus (TRSV) (1, 2, 3, 4, 5, 6). Prevalence of a single virus in limited areas depends on the presence of insect vectors and on the abundance of primary inoculum.

In the Lower Rio Grande Valley of Texas watermelons, cantaloupes, and Honey Dews are concentrated in widely separated areas (approximately 100 miles distance), although limited acreages of each, as well as squash and cucumber, may be found anywhere.

Most cucurbits are planted in January-February and harvest usually terminates in May or June. A limited acreage of squash, cucumber, and cantaloupe is frequently planted in September for fall production, so that virus diseases may be studied in the field during these two periods.

Surveys during two spring and one fall seasons of 1959-60 of the viruses affecting cucumber, squash, cantaloupe, Honey Dew, and watermelon in the Lower Rio Grande Valley of Texas indicate a different virus complex from those reported on cucurbits from California (3), Arizona (3, 4), Florida (1), and Wisconsin (5). This is a report of preliminary assays carried out at Weslaco, Texas.

METHODS AND MATERIALS

Terminal growth removed from diseased field-grown plants was used as a source of inoculum for indexing to selected test plants. Each virus sample was triturated in a mortar and the juice extract was inoculated to a series of test plants which gave a differential reaction to each virus (2, 3, 5). Most inoculations were made in a greenhouse; during warmer temperatures some studies were conducted in a screenhouse. Cheesecloth swabs were used to apply inoculum to test plants previously powdered with carborundum (No. 320).

The reactions of different test plants to known isolates of CMV (from G. S. Pound, Wisconsin), SqMV (from J. H. Freitag, California), and WMV (from C. W. Anderson, Florida) were compared with the reactions of the cucurbit virus samples from the Rio Grande Valley. Symptoms produced by TRSV were reported previously (6).

Test plants consisted of Vigna sinensis (cowpea vars. Blackeye, Black (Sill and Walker strain)), Phaseolus lunatus (lima bean var. Thoroughgreen), Cucumis sativus (cucumber var. Palomar), Cucumis melo (cantaloupe var. Rio Gold), Citrullus vulgaris (watermelon var. Charleston Grey), Cucurbita pepo (squash var. Prolific Straightneck), Zinnia elegans (zinnia var. Lilliput), and Nicotiana glutinosa. From the reactions of these plants the virus content of each sample was identified: as a strain of CMV from local lesions produced on inoculated primary leaves of Blackeye cowpea or systemic mottle in trifoliate leaves of Black cowpea (Sill and Walker strain, susceptible to systemic infection) (7), systemic mottle in zinnia and N. glutinosa, severe systemic yellow mottle in cucumber, cantaloupe, and squash, necrotic spotting

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on inoculated leaves of watermelon; as TRSV from necrotic lesions produced on inoculated primary leaves and necrosis of terminals becoming lethal in cowpea, large necrotic blotches and vein necrosis on inoculated primary leaves and occasional systemic necrosis in lima bean (6), very mild to no reaction with recovery in zinnia and N. glutinosa, chlorotic stippling, ring spotting, and mild mottle in cucumber, cantaloupe, watermelon, and squash; as a strain of WMV from no reaction in cowpea, lima bean, zinnia, or N. glutinosa, pronounced mottle in cucumber and cantaloupe, systemic mottle, stunting, and leaf distortion in watermelon and severe filiform leaf distortion, mottle and blistering in squash; as a strain of SqMV from no reaction in cowpea, lima bean, zinnia, N. glutinosa, or watermelon, mild vein-banding and mottle with recovery in cucumber and cantaloupe, severe mottle with green blisters and leaf distortion in squash.

RESULTS AND CONCLUSIONS

Results from these assays are summarized in Table 1. Nearly all virus isolates from cucumber, cantaloupe, and Honey Dew caused reddish local lesions on inoculated primary leaves of Blackeye cowpea followed by systemic lethal necrosis within 3 to 7 days. Parallel inoculations to zinnia, N. glutinosa, and Sill and Walker strain of Black cowpea showed this virus to be TRSV. TRSV symptoms were general and widespread in watermelon; certain fields were 100% infected. The virus was transferred with difficulty from watermelon foliage to most test plants (6). Symptoms of WMV were seldom observed in watermelon and the virus did not appear to spread from primary infections. WMV was recovered from a few plants of other cucurbits but rarely from watermelon. WMV, SqMV, and TRSV were isolated either alone or in combination from squash. SqMV was recovered only from squash. Most isolates contained TRSV but failure of an isolate to react on cowpea and watermelon showed the presence of SqMV and the absence of TRSV.

Table 1. Relative prevalence of cucurbit viruses in the Lower Rio Grande Valley of Texas during two spring and one fall seasons of 1959-60.

Virus host plant	No. virus samples tested	Virus in host plant as indicated by indexing tests			
		CMV	TRSV	WMV	SqMV
Watermelon	15		13	2	
Squash	26		16	2	10
Cucumber	13		12	1	
Cantaloupe	86	2	83	13	
Total	140	2	124	18	10

Symptoms of TRSV were widespread in young plants of all cucurbits but became masked with marked recovery in older plants. Symptoms persisted longer in squash with severe stunting. Isolates from older plants of squash, cantaloupe, and Honey Dew caused not only the usual symptoms of TRSV on test plants, but frequently caused vein-clearing, blistering, and leaf distortion in inoculated squash plants. Watermelon plants inoculated with these virus isolates, after showing the usual chlorotic stipple symptoms of TRSV, were severely stunted, distorted, and frequently showed mosaic mottle indicating the presence of WMV also. Symptoms of the combined viruses in watermelon were more severe than those of either virus alone. Although this condition has not been observed on watermelon in the field, it has been duplicated several times after inoculations with the respective viruses TRSV and WMV.

The paucity of CMV is unexplained. This virus was recovered from only two of 140 cucurbit plants assayed and occurred in association with TRSV. Since both viruses cause somewhat similar reactions in most test plants, the persistence of mosaic mottle in zinnia and N. glutinosa was the best indicator of the presence of CMV. When CMV and TRSV were combined, inoculation on Black cowpea caused local necrotic lesions on primary leaves followed by systemic necrosis without mosaic mottle, indicating the presence of TRSV which was lethal in cowpea before the expression of CMV symptoms.

The only suggested host-range method of differentiating between SqMV and WMV is the failure of SqMV to react systemically in watermelon (2, 3, 5). The presence of TRSV in many cucurbit virus isolates caused systemic chlorotic stippling in watermelon 5 to 7 days after inoculation, with eventual recovery. Failure of infected plants to recover and ensuing severe stunt-

ing and leaf distortion indicated the presence of WMV. Symptoms of CMV, TRSV, SqMV, and WMV usually were differentiated in squash seedlings inoculated in the cotyledon stage. Also, the incubation period before distinct symptoms were expressed was different for each virus: CMV 8-14 days, TRSV 3-7 days, SqMV 10-14 days, WMV 12-16 days.

TRSV was the prevalent virus in all cucurbit fields surveyed during the spring in the Lower Rio Grande Valley. CMV and WMV were recovered in the fall. The rare occurrence of CMV indicates that the vector or a reservoir of that virus is lacking in the principal cucurbit culture districts of the Lower Rio Grande Valley. The prevalence of TRSV and the rare occurrence of other cucurbit viruses indicate that the virus complex in the Lower Rio Grande Valley of Texas is very different from the cucurbit virus complexes reported elsewhere. Such differences in prevalence of the several cucurbit viruses may be associated with differences in relative abundance of vectors, reservoir hosts, both, or other unknown factors.

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OCCURRENCE OF CERTAIN PLANT DISEASES IN KENTUCKY IN 1960[1]

E. M. Johnson, R. A. Chapman, W. D. Valleau, and George A. Everette

High rainfall and low temperatures delayed the preparation and sowing of tobacco beds throughout the State. Following sowing time a statewide drouth occurred, lasting for most of the plant bed season. Because watering of beds was delayed too long or was poorly done, plant bed failures were numerous. This resulted in many very late crops with resultant poor cure and in increased movement of plants from farm to farm and from area to area.

TOBACCO PLANT BED DISEASES

Fertilizer injury. In dry seasons, as occurred in 1959 and 1960, excess fertilization results in an accumulation of salts at or near the soil surface, which causes small plants 1/4 to 2 inches across to turn yellow and die. Such plants when touched break off at the surface of the ground. This condition was widespread in the State and contributed to plant bed failures. Many growers, believing the yellowing and poor growth to be due to lack of nitrogen, aggravated the condition by applying large amounts of soluble nitrogen fertilizers.

Wildfire (Pseudomonas tabaci (Wolf & Foster) Stevens) and angular leaf spot (Pseudomonas angulata (Fromme & Murray) Holland) were present in a few plant beds but neither caused appreciable damage. Angular leaf spot was identified in several beds of Burley 21.

Anthracnose (Colletotrichum destructivum O'Gara) was present in some plant beds throughout the State. Losses in Calloway and Muhlenburg counties were often high, and several plant beds in each of these counties were destroyed.

Blue mold (Peronospora tabacina Adam) appeared in a few beds in widely separated areas at or after setting time. Sporulation was sparse or entirely absent.

Cold injury was present in most beds throughout the State but caused no damage.

FIELD DISEASES OF TOBACCO

Pythium soft rot (Pythium spp.) occurred in tobacco fields over all the State soon after plants were set. There was a great deal more than in 1959. Some growers were forced to reset entire crops. As temperatures rose no further trouble developed.

Black shank (Phytophthora parasitica Dast. var nicotianae (Breda de Haan) Tucker) occurred on many farms with no previous history of the disease. In one neighborhood where there was no previous record of black shank the disease is believed to have been brought in on plants from another area where black shank was known to occur. Every farmer in this neighborhood who set his crop with plants from the same black shank-infested area had the disease whereas those in the same neighborhood who set their own plants had no black shank. Black shank is now appearing on many of the larger farms in the central area of the State often in fields next to well travelled highways. Burley 37 tested in black shank-infested fields with Burley 11A and 11B appears to be more resistant than either.

Manganese toxicity seems to be increasing. Affected plants grow slowly for some time following setting, turn light green or yellow between the larger veins and gradually develop numerous small dead spots, especially on the older leaves. The injury occurs in areas of fields where the soil is very acid (pH 4.5 to 5.2). Chemical analyses of affected plants from 20 locations showed parts per million of manganese from 1125 to 6880. Normal plants contain about 400 ppm of manganese or less.

Frogeye (Cercospora apii Fres.) [= C. nicotianae Ell. & Ev.] was less prevalent than last year; however, the green spot stage of the disease was more prevalent, particularly on late maturing crops.

Etch, an aphid-transmitted virus disease, was reported more frequently than usual. In some burley fields the percentage of affected plants was high. Etch adversely affects both yield and quality of burley tobacco. If etch continues to increase at the present rate it may seriously affect tobacco production.

Wildfire was of minor importance but angular leaf spot appears to be increasing. Fields of Burley 21, resistant to wildfire, were observed where nearly all leaves on every plant were affected by angular leaf spot and the damage appeared great.

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[1] The investigation reported in this paper is in connection with a project of the Kentucky Agricultural Experiment Station and is published with approval of the Director.

Black root rot (Thielaviopsis basicola (Berk. & Br.) Ferr.) again caused damage in all areas of the State, particularly in fields used each year for tobacco. Temperatures, especially those at night, were low well into July. The most popular burley varieties, Ky 16, Burley 21 and Ky 35, have low resistance to black root rot and should be replaced by the newer highly resistant varieties except in rotations.

Bacterial black stalk, after an absence of several years, appeared late in the season in six widely separated areas. All leaves of affected plants were yellow and the bark was blackened from the ground line sometimes for a distance of 2 1/2 feet. Only a few plants in a field were affected.

ELM

Dutch elm disease (Ceratocystis ulmi (Buis.) Moreau) was identified by isolation from elms in Fayette, Adair, Bourbon, Boyle, Mason and Shelby counties.

APPLE

Fireblight (Erwinia amylovora (Burr.) Winslow et al.) was present in greater amounts in apples and pears than for many years. In neglected backyard gardens pear trees often had nearly all of the leaves affected. Neglected apple trees were only slightly less affected. The disease was present and conspicuous on flowering crabs.

KENTUCKY AGRICULTURAL EXPERIMENT STATION, LEXINGTON

EFFECTIVENESS OF CERTAIN PROTECTANT FUNGICIDES FOR CONTROLLING PECAN SCAB IN OKLAHOMA DURING 1959

George L. Barnes[1]

Abstract

Thirteen protectant fungicide preparations were tested on pecan trees of the Western variety in Oklahoma for control of scab on foliage and nuts during the severe scab season of 1959. The materials were applied with a hydraulic spray machine on a 2-week application schedule. Excellent control was provided by zineb fungicides (Parzate, Dithane Z-78, tank-mix zineb) and by Dyrene, Cyprex, and Bordeaux mixture. Ziram fungicides (Zerlate, Niacide Z, tank-mix ziram), and Amobam and Dibam A were ineffective. Bordeaux applications, though effective, resulted in an aphid build-up and in some russetting of nut shucks. Zineb is recommended for scab control in Oklahoma.

INTRODUCTION

Pecan scab, caused by the fungus Fusicladium effusum Wint., limits the production of nuts throughout most of the pecan belt, and control of this disease is of great importance to the industry. The disease causes great losses in localities where rains are frequent and long periods of high humidity prevail. When trees are not sprayed with effective fungicides during wet seasons, nuts of most of the improved varieties and some of the nuts of the seedling or native trees are lost because of scab infections. Proper timing of applications of effective fungicides and strict adherence to good sanitation practices, such as removing excess trees and shallow plowing under of diseased plant debris, will greatly facilitate control of scab as well as of other diseases.

A wide variety of fungicidal materials have been tested for control of pecan scab by various workers. The results varied with wet and dry seasons. Low-lime Bordeaux mixture alone or a split schedule with Bordeaux followed by zinc ethylene bisdithiocarbamate (zineb) or zinc dimethyldithiocarbamate (ziram) is generally recommended in the southeastern States (4). In the southwestern States, Bordeaux mixture causes injury during hot, dry weather and also causes aphid and mite populations to increase after its use (11). The disadvantages of using Bordeaux have prompted a search for more suitable materials. Zineb has been found to be very effective in both wet and dry seasons (1). Zineb is the currently recommended fungicide for scab control in Texas and Oklahoma (1, 11). Recently, two other types of organic fungicides, n-dodecylguanidine acetate (dodine) (Cyprex[2]) and 2,4-dichloro-6-(o-chloroanilino)-s-triazine (Dyrene[2]), have been found to be very effective during severe scab seasons (1, 3).

The presently reported test was designed to obtain information in several categories. One objective was to obtain additional data on the relative effectiveness of dodine and Dyrene. Another objective was to evaluate the effectiveness of tank-mix preparations of zineb and ziram because tank-mix preparations are more economical to use than commercial preparations. The third objective was to determine the effectiveness of two new dithiocarbamate fungicides, Amobam and Dibam A, which had been found by the author to be toxic to F. effusum in laboratory tests.

MATERIALS AND METHODS

During 1959, 13 fungicide treatments were field-tested to determine their effectiveness in controlling scab on pecan trees of the Western variety. Commonly used scab control fungicides, low-lime Bordeaux mixture, zineb (Parzate and Dithane Z-78), and ziram (Zerlate) were included as well as four new experimental fungicides, Cyprex, Dyrene, Amobam, and Dibam A, and tank-mix preparations of zineb and ziram (Tables 1 and 2).

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[2]Mention of a trade name does not constitute a guaranty or warranty of the product named and does not signify that it is approved to the exclusion of other comparable products.

Table 1. Active ingredients and manufacturers of commercial fungicides used in pecan scab control trials in Oklahoma during 1959.

Fungicide	Active ingredients	Manufacturer
Parzate	65% zineb	E. I. du Pont de Nemours & Co., Inc.
Dithane Z-78	65% zineb	Rohm & Haas Co.
Zerlate	76% ziram	E. I. du Pont de Nemours & Co., Inc.
Niacide Z	59.8% ziram + 5.2% zinc mercaptobenzothiazole	Niagara Chemical Division, Food Machinery & Chem. Corp.
Dyrene	50% 2,4-dichloro-6-(o-chloroanilino)-s-triazine	Chemagro Corporation
Cyprex	65% dodine	American Cyanamid Co.
Amobam	42% diammonium ethylene bisdithiocarbamate	Roberts Chemicals, Inc.
Dibam A	41.7% ammonium dimethyl-dithiocarbamate	Roberts Chemicals, Inc.

Table 2. Effectiveness of certain protectant fungicides for control of scab on Western pecan trees in Oklahoma during 1959.

Treatment[a]		Degree of scab control (foliage and nuts)[b]	Average number of nuts per pound[c]
Fungicide	Rate/100 gallons of spray		
Parzate	3 lb.	excellent	58
Parzate + Standard Superla summer oil	3 lb. + 1 pt.	excellent	67
Dithane Z-78	3 lb.	excellent	57
Tank mix zineb-nabam + $ZnSO_4$	2 qt. + 1.25 lb.	excellent	74
Zerlate	3 lb.	very poor	112[d]
Niacide Z	4 lb.	very poor	124[d]
Tank mix ziram-Dibam A + $ZnSO_4$	1 qt. + 1.25 lb.	poor	93[d]
Bordeaux mixture-$CuSO_4$ + lime	6 lb. + 2 lb.	excellent	78
Dyrene	4 lb.	excellent	64
Cyprex	1 lb.	excellent	84
Cyprex + Standard Superla summer oil	1 lb. + 1 pt.	excellent	62
Dibam A	1 qt.	very poor	146[d]
Amobam	1 qt.	very poor	108[d]
None	---	---	215[e]

[a]Two-week schedule. Nine applications.
[b]Determined in October.
[c]Nuts collected, weighed, and counted on November 5 and 6.
[d]Nuts only partially filled and not marketable.
[e]Unfilled nuts with tightly adhering, scab-blackened shucks.

Spray applications were made with a conventional hydraulic spray machine operating at a pressure of 600 psi. The trees were individually circled and approximately 20 gallons of spray material was applied to each tree on each of nine spraying dates. A 2-week application schedule was followed for two reasons: 1) There is presumptive evidence that the incubation period of the fungus averages about 10 days in the field (6). 2) The effectiveness of pesticide deposits may rapidly diminish as a result of erosion by rain and dew, brushing of foliage, thinning and breaking of the deposit by tissue growth (7), and possible decomposition by high temperatures and strong sunlight (10). An effort was made to assure complete coverage because poor coverage can result in poor control by even the best of fungicides. Randomized single tree plots of

27-year-old trees were replicated five times for each treatment.

Rain and dew occurred frequently during the entire season and many days and nights were favorable for infection of foliage and nuts. Slightly over 23 inches of rain fell in the grove during 41 rainy days (0.01 inch and over) between May 1 and September 30. Non-sprayed control trees became severely scabbed. Cole (2) and Large (9) reported great difficulty in controlling scab under such conditions.

Because scab-infected nuts do not fill properly, the weight of nuts from trees in fungicide plots can be used to determine the degree of scab control (5, Table 4). This method was used to assess the effectiveness of the fungicides in the 1959 test.

RESULTS AND CONCLUSIONS

Of the fungicidal treatments evaluated during 1959, the zineb fungicides (Parzate, Dithane Z-78, tank-mix zineb) and Dyrene, Cyprex, and low-lime Bordeaux provided excellent control of scab (Table 2). The ziram fungicides (Zerlate, Niacide Z, tank-mix ziram) and Amobam and Dibam A were ineffective. The nuts from trees sprayed with the latter materials were only partially filled and were not marketable. Bordeaux mixture, though effective, resulted in a buildup in aphid populations and in some russetting of nut shucks.

Zineb materials, Dyrene, and Cyprex warrant further evaluation as protectant fungicides for scab prevention in Oklahoma. Zineb is recommended for use in Oklahoma because of its effectiveness and availability. Of additional interest are the reports that zineb suppresses pecan mite and aphid populations (8, 12). Although Dyrene and Cyprex are efficient scab-control fungicides, they cannot be recommended for grower use yet because residue tolerances have not been established by the Food and Drug Administration for these fungicides on pecans (as of December 1, 1960).

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SEPTOCYLINDRIUM AROMATICUM ON SWEET FLAG

Anna E. Jenkins and Ahmés P. Viégas[1,2]

A fungus not often collected in recent years in the United States is Septocylindrium aromaticum Sacc. (Cylindrosporium acori Pk.) on sweet flag (Acorus calamus). In 1937 the writers collected this fungus at Walton, N. Y. (Fig. 1, A and B), and in 1940 W. W. Diehl found it abundant on this plant growing on Theodore Island (vicinity Washington, D. C.), Potomac River. In the National Fungus Collections the several other specimens from the United States are from Cortland (1923) and E. of Forest Home (1916), N. Y.; New Brunswick, N. J. (1893) and Newark, Del. (1907). Additional distribution represented by specimens in the herbarium, Department of Plant Pathology, Cornell University, is as follows: East Granby, Conn. (1910); Hudson Falls (1920), Labrador Lake (1923) and North Spencer (1916), N. Y., besides Stockton, Kans. (1913). In the herbarium, N. Y. State Museum, are three specimens from New York, namely Albia (1924), New Haven (1921) and Sand Lake (no date, coll. Chas. H. Peck, doubtless the type of his C. acori), with one from Madison, Wis. (1915).

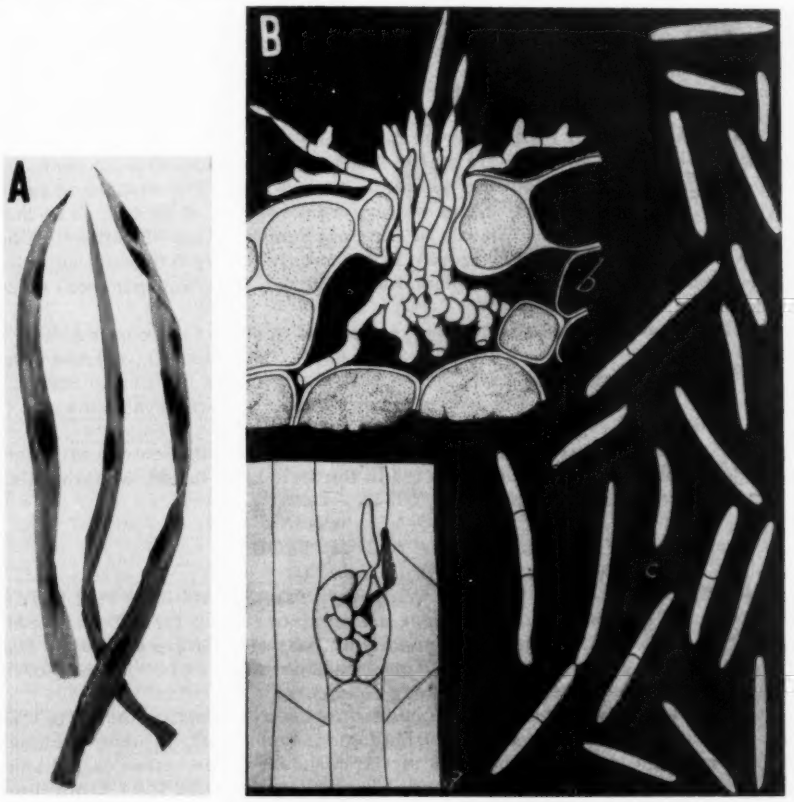

FIGURE 1. Septocylindrium aromaticum on leaves of sweet flag, Walton, N.Y. A -- X about 3/4. B -- a-c, drawings by Viégas.

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[2] We are grateful to the curators of the herbaria mentioned for the privilege of consulting the specimens of Septocylindrium aromaticum in those collections.

THE SUSCEPTIBILITY OF SAFFLOWER VARIETIES AND SPECIES TO SEVERAL FOLIAGE DISEASES IN ISRAEL

Amram Ashri[1]

Abstract

The reaction of 39 varieties and lines of safflower and of 6 wild Carthamus species to Puccinia carthami, Ramularia, Cercospora, Alternaria, or Stemphylium, powdery mildew and "white spots" is described. Symptoms of the diseases are given as well. It is suggested that the P. carthami race or races found in Israel do not correspond to the four races established in the United States of America.

The author started experiments with safflower in Israel in 1959, but they were preliminary only. The more extensive 1960 plots afforded an opportunity to study the reaction of many lines to several diseases and to describe the pathogens in detail. The latter subject will be treated separately (8). The purpose of this article is to discuss the susceptibility of the different varieties and species to the diseases, for the benefit of breeders and pathologists.

LITERATURE REVIEW

Minz (7) described the two safflower rust pathogens prevalent in Israel, namely P. carthami Corda and P. verruca Thuem. He stated that both were found also on herbarium specimens of wild Carthamus species, but did not name the species. The disease caused by Ramularia carthamicola Darpoux is fairly common in certain regions of Israel, as is the leaf spotting caused by Alternaria carthami Chowd. or Stemphylium species. Powdery mildew (Erysiphe cichoracearum f. carthami Milovtzova) is found only rarely (personal communication, Minz). Cercospora has hitherto not been found on safflower in Israel (personal communication, Solel).

The susceptibility of various safflower varieties and species to one or more of these diseases was reported by several authors (2, 3, 4, 5, 6, 9). Thomas (10, 11) has established the prevalence of at least four pathogenic races of P. carthami in the United States.

Working at Davis, California, the author found that Carthamus oxyacantha and C. palaestinus were badly rusted. Several other species, C. alexandrinus, C. glaucus, C. tenuis, C. syriacus, C. lanatus and C. baeticus, were free from rust. Furthermore, all attempts to inoculate them artificially with spores collected in the field have failed (McCain, University of California, Davis, personal communication, 1957).

MATERIALS AND METHODS

The preliminary adaptation test with 39 cultivated varieties and lines (Table 1) was planted on March 30, 1960 at Rehovot, Israel. A large uniform nursery of foreign and local wild species and ecotypes (Table 2) was sown adjacently as part of a different study. The cultivated varieties were planted in 3-row plots, without replications. Rows of N-10 and Australian Spineless were scattered throughout the nursery as checks.

C. palaestinus was represented by the line used in the cytogenetic studies in California (1) and by local collections. There were two morphological types of C. nitidus, a smooth, typical one and a hairy, woolly one. C. glaucus was the large-headed type, often called anatolicus. C. tenuis was represented by local collections. The species labeled C. ? from the Upper Galilee is very similar to the C. syriacus studied in California (1). It also resembles closely the specimens of C. alexandrinus and C. dentatus seen at the Kew Gardens Herbarium.

All seeds were treated with tetramethylthiuram disulfide (thiram) (Arasan) before planting. The plots were irrigated regularly with overhead sprinklers. No artificial infection was practiced. It should be noted that at Rehovot the latter part of the growing season was hot and humid.

RESULTS AND DISCUSSION

Rust infection became serious by mid-May. The other diseases became widespread 3 to 4 weeks later when flowering began. Only a short description of their symptoms will be given here.

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Table 1. Susceptibility of safflower varieties to three leaf diseases, natural infection.

Variety[a]	Accession number	Country of origin	Susceptibility[b] to: Rust	Ramularia	Cercospora
No. 34	1-59-3	India (Nagpur)	2	rare	1
N.P. 1	do. -27	do. (New Delhi)	2	0	2
N.P. 5	do. -28	do. do.	2	0	2
N.P. 8	do. -29	do. do.	2	rare	2
N.P. 29	do. -4	do. do.	2	rare	1
N.P. 30	do. -30	do. do.	3	1	2
N.P. 32	do. -5	do. do.	2	0	1
U.C. 57-146	1-60-11	do.	2	0	2
23(1)B	1-59-6	Australia	1	0	1
23(3)-5	do. -7	do.	1	rare	1
48-89	do. -8	do.	2	1	1
55-1	do. -9	do.	2	1	1
R.R. 340-53	do. -10	do.	2	rare	rare
2B	do. -11	do.	2	2	2
U.C. 56-15	1-60-5	do.	2	0	2
Nebraska 6	1-59-12	Nebraska	2	0	2
Nebraska 852	do. -14	do.	2	0	2
Unknown[c]	do. -15	do.	2	1	2
Nebraska 10	do. -16	California	2	1	2
Australian Spineless	do. -19	do.	2	1	2
Pacific 1	do. -20	do.	2	1	2
Pacific 2	1-59-17	do.	2	1	2
Pacific 7	do. -21	do.	2	1	2
Western Oilseeds 14	do. -18	do.	2	1	2
Local	--	Israel	2	2	2
do.[d]	1-59-24	Spain	2	0	1
do.	do. -25	Morocco	2	1	1
Tashkentskii	do. -31	Bulgaria	2	3	2
Nebraska 8	do. -32	do.	2	3	2
P.I. 228,283	1-60-1	Iran	3	2	2
U.C. 55-43	do. -3	Sudan	2	1	2
P.I. 209,295	do. -4	Kenya	2	1	2
U.C. 59-576-1	do. -7	Arizona	2	1	2

[a] U.C. indicates a breeding line from the University of California at Davis. P.I. numbers are U. S. introductions.

[b] 0 indicates no infection; 1, trace infection, few leaves diseased, only a small part of the leaf infected; 2, heavy infection, many leaves diseased, a bigger part of the leaf infected; 3, severe infection, most leaves diseased, a bigger part of the leaf infected.

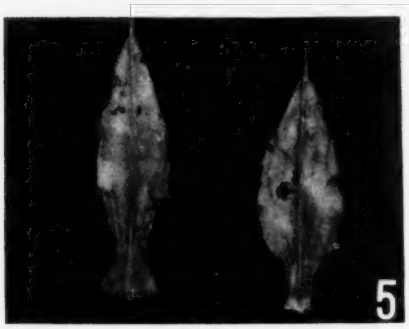
[c] Received unnamed from Israel's Ministry of Commerce and Industry.

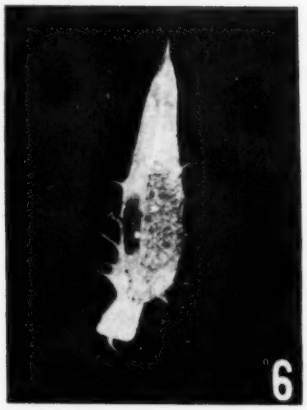
[d] One plant only.

Table 2. The susceptibility of wild Carthamus species to four leaf diseases.

Species	Number of rows	Country of origin	Susceptibility[a] to: Rust	Ramularia	Cercospora	Powdery mildew
C. oxyacantha	2	India	1	0	0	0
C. palaestinus	32	Israel	3	1	2	x
C. nitidus	8	Israel	1	0	0	0
C. glaucus	35	Israel	2	0	1	x
C. tenuis	26	Israel	2	0	0	0
C. ?	19	Israel	2	0	0	0

[a] 0 to 3 as in Table 1. For powdery mildew x indicates presence. No estimates of its severity were undertaken.





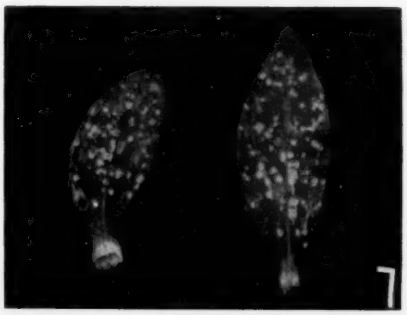


FIGURE 1. Left to right: P. carthami on the underside of the leaves of C. oxyacantha, C. palaestinus, W.O. 14 and N-10.

FIGURE 2. Left to right: P. carthami on underside of rosette leaves of C. glaucus and C. tenuis, and C. nitidus stem leaf. Note smaller pustules and chlorotic rims.

FIGURE 3. Ramularia leaf spots on both sides of outer involucral bracts of local safflower. Small dark dots are rust pustules.

FIGURE 4. Cercospora on upper (left) and lower (right) side of leaves of the variety 2B.

FIGURE 5. Powdery mildew on lower side of C. palaestinus leaves. Dark spots are rust pustules.

FIGURE 6. A leaf of N.P. 29 showing an Alternaria (or Stemphylium) spot (left) and Cercospora (right).

FIGURE 7. "White spots" on upper side of leaves of Australian Spineless. Dark dots are rust pustules.

Rust: Only P. carthami was found. Pustules varied in size and the degree of scattering on the leaves. They were present predominantly on the underside of the leaves and had the typical chocolate-brown color. The epidermis on the pustules was ruptured (Fig. 1). Both rosette and stem leaves of the wild species were infected. Generally, on these, the pustules were smaller and more scattered. A chlorotic, pale rim surrounded the pustules. (Fig. 2).

Ramularia: The leaf spots caused by this pathogen are round and regular. They are characterized by a whitish dense mass of conidiophores and conidia in the center which reflect the light when held at a proper angle. The brown color of the dry spots is especially noticeable in the margins. Their size varies and may reach a radius of more than 5 mm. The spots are found on both sides of the leaf. (Fig. 3).

Cercospora: Leaf spots caused by this pathogen are usually regular and round, and may reach a radius of 1 cm. They can be easily identified by the dull, dark-grey spore-bearing conidiophores which appear in concentric rings. (Figs. 4 and 6).

Powdery Mildew: Symptoms are similar to those encountered in other crops. (Fig. 5).

Alternaria or Stemphylium: Unfortunately it could not be determined which of the pathogens caused the primary infection. The leaf lesions were dark brown, slightly sunken, and irregular in shape. The larger spots measured about 1 cm across. (Fig. 6).

Another abnormal spotting could not be identified. Small, white round spots developed on the upper side of the leaves and outer involucral bracts by late April. Virus, bacteria, fungi and insect damage were ruled out. It may be a physiological, or a nutritional disorder. On the underside of the leaves there were small, light brown sunken spots, but not always corresponding to the locations of the white spots. (Fig. 7).

The disease did not appear to be antagonistic. Rust, Ramularia, Cercospora, Alternaria (or Stemphylium) and "white spots" were often found on the same leaf. Powdery mildew was rather uncommon, still, leaves infected with it and rust were found.

The susceptibility of the various varieties and lines to three diseases is summarized in Table 1. In addition to these lines there were four thin-hulled lines derived from Rubis' mutants. Since their reaction was identical to that of U.C. 59-576-1 which has the same history, they were not included in the table. Duplicate varieties, obtained from different sources were entered only once. The susceptibility of the wild species to four diseases is presented in Table 2. In all cases, susceptibility estimates were based on the degree of infection of the population of each variety or species.

Not even one variety was resistant to P. carthami (Table 1). A trace of infection was found only on two Australian varieties, 23(1)B and 23(3)-5. To be sure, these varieties were early. However, it would appear that the severity of the infection was not related to the earliness of the lines. Of the severly infected, N.P. 30 was very early while P.I. 228,283 was late. All wild species included in the test were rusted (Table 2).

The many varieties and lines which were free of Ramularia (Table 1) may be truly resistant, but it is very likely that there were many escapes. It appears that most of the varieties which were on the western side of the plot were in the "0" or "rare" group, including N-10 which was infected elsewhere in the nursery. Of the wild species, Ramularia was found on C. palaestinus only (Table 2). The early C. oxyacantha may have escaped infection. The other species, though, were much later than the cultivated materials. Thus, it is presumed that they are resistant to this disease.

Cercospora inoculum was well distributed throughout the nursery. Once it appeared it spread very quickly. All the varieties were infected (Table 1). However, R.R. 340-53 proved nearly resistant. Four wild species were not diseased (Table 2). C. oxyacantha may have escaped infection because of its earliness. This suspicion is strengthened by the fact that it is infected in India (6). Thus, sources of resistance to the disease may be hard to find among the cultivated varieties and the related wild species.

Alternaria (or Stemphylium) leaf spots were found on all varieties except for the Indian low-iodine line, U.C. 57-146. The most sensitive variety was P.I. 228,283. The leaf spots were not present on C. glaucus, C. tenuis and C. ? which are only distantly related to the cultivated species. The other wild species were infected.

Powdery mildew occurred in a few localized spots in the nursery. All that can be said here is that 55-1, C. palaestinus and C. glaucus were susceptible.

All varieties, lines and wild species had the "white spots."

Thomas (10, 11) has identified several pathogenic races of P. carthami in the United States. His differentials were not included in the 1960 nursery. However, on the basis of the reaction of the wild species, it would appear that additional race or races are present in Israel. Thomas (11) found that races 2 and 4 were present at Davis in 1957 and 1958. In 1957

the author did not find there any rust on plants of C. glaucus and C. tenuis which originated in Israel. Thus, they may be resistant to these races. In nearby Woodland, California, Thomas (11) found races 2 and 3. Since personnel from Davis and Woodland visited each other's plots it is reasonable to assume that race 3 inoculum could have reached the experimental nursery at Davis. On these admittedly tenuous grounds, it is assumed that C. glaucus and C. tenuis were resistant to race 3 as well. Finally, race 1 gives only a "1" reaction on Pacific 1, Pacific 2 and W.O. 14. These species were well rusted here. Thus, the race which infected the cultivated and wild species was not race 1. On this evidence it is suggested that the P. carthami race found at the nursery is a new race which has not hitherto been described. Thomas' differentials will be planted next season in order to test the above suggestion and to establish the identity of the local race or races.

A test with artificial inoculation is planned for the following season. Many additional varieties will be screened for resistance to these diseases.

ACKNOWLEDGMENTS

The author gratefully acknowledges the help of Mr. Zvi Solel of the Agricultural Experiment Station, Beit-Dagan, in the identification of the various diseases. The help of Dr. Y. Kostrinsky of the above station, and of Dr. P. F. Knowles, University of California, Davis, who supplied the seeds of many lines, is sincerely appreciated.

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GYMNOSPORANGIUM FUSCUM FOUND ON PEARS IN CALIFORNIA

Arthur H. McCain[1]

The aecial stage of Gymnosporangium fuscum DC. (=G. sabinae Dicks. ex Wint.) was found on pear, Pyrus communis, growing on home grounds near Lafayette, Contra Costa County, California. This is the first report of the disease from California. No swellings were evident on nearby Juniperus species. A search will be made for the telial stage in the spring.

It is believed that this is the first report of the disease from the United States. Saccardo's (3) record of the disease in North America appears doubtful and may have been based on Farlow's (1) report of G. fuscum var. globosum (=G. globosum Farlow). The rust was intercepted by the U. S. D. A. Plant Quarantine Branch on Juniperus chinensis var. pfitzeriana arriving by air from Germany or the Netherlands between July 1, 1952 and June 30, 1953 (2). It is possible that the disease was introduced into California by infected junipers.

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SUGAR BEET NEMATODE FOUND IN NEW YORK STATE

W. F. Mai

Large numbers of immature females of the sugar beet nematode, Heterodera schachtii Schmidt, 1871, were found on the roots of unthrifty red beets, Beta vulgaris, growing in muck soil near Syracuse, New York.

In addition, larvae and cysts of this nematode were recovered from soil samples collected from this field. In a large spot in the field a high percentage of the plants were dead and the growth of the remaining plants was reduced markedly. It is believed that this is the first record of the occurrence of the sugar beet nematode in New York State.

The plant and soil samples were collected by Mr. H. B. Trull, Assistant Agricultural Agent of Onondaga County.

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EXTENSION OF RANGE AND A NEW HOST FOR CRISTULARIELLA PYRAMIDALIS

Charles R. Berry and George E. Thompson[1]

Cristulariella pyramidalis was described as a new species by Waterman and Marshall[2] in 1947. They reported it on living leaves of Acer negundo, A. saccharum, and A. pseudoplatanus in Connecticut, New York, and Pennsylvania.

In October 1960, C. pyramidalis was found associated with premature defoliation of A. platanoides in Asheville, North Carolina. Several trees in one location, ranging in height from 15 to 50 feet, were infected and three trees had been largely defoliated. Nearby uninfected or lightly infected trees had not shed their leaves by November 1. Adjacent trees of species A. saccharum and A. saccharinum were apparently free of infection.

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[2]Waterman, A. M., and R. P. Marshall. 1947. A new species of Cristulariella associated with a leaf spot of maple. Mycologia 39: 690-698.

CERATOCYSTIS ULMI AND ZINC CHLORIDE EXPERIMENT

George E. Hafstad and William C. Lueschow

During the summer of 1960 many reports and requests concerning the use of zinc chloride or zinc-coated nails for the prevention and cure of Dutch elm disease were received. Recommended treatment for the control of Dutch elm disease consisted either of drilling a hole into the tree and filling it with a solution of zinc chloride or driving zinc-coated nails at 2-inch intervals around the circumference of the tree. Because of the many requests as to the value of zinc chloride, it was thought worthwhile to check its toxicity to the Dutch elm disease fungus, Ceratocystis ulmi, in the laboratory.

Solutions of 10, 25, 50, 100, 250 and 500 ppm of zinc chloride were made in volumes of 150 cc. Into each of these volumes approximately 3.5 grams of Difco PDA was added. After sterilization, the PDA was poured into Petri dishes.

Transfers from a stock culture of Ceratocystis ulmi were made in triplicate. Checks were included. All were incubated for a week at 68° F. After 7 days, the plates were observed; excellent growth had occurred in the entire series. In fact, the growth on some of the 10, 25, and 50 ppm plates was more profuse than that in the checks. This is thought to be the result of some variation in, or the amount of, transfer material.

There was a slight reduction in growth in the 250 and 500 ppm plates but not sufficient to be significant. From these observations it may be stated that in concentrations up to 500 ppm zinc chloride did not produce any inhibitory effect on the growth of C. ulmi under laboratory conditions.

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SEED-BORNE CERCOSPORA ON SAFFLOWER

Amram Ashri[1]

In a companion paper[2] the susceptibility of various safflower varieties and species to Cercospora carthami (H. & P. Syd.) Sund. & Ramak. has been described. The disease has not hitherto occurred in Israel and furthermore in the 1960 season it appeared only in the author's nursery which included many introductions. It is very likely, then, that the disease has been introduced with the seeds of one of the introductions. This has several implications:

The first reflects upon the value of phytosanitary certificates, since all introductions came with them. It would be advisable to tighten up inspection at the source, where personnel are aware of the prevailing diseases. The recipient should be advised which diseases infect the crop.

Secondly, it is apparent that Cercospora is seed-borne also in safflower. Thus, methods of seed treatments should be investigated.

Finally, treatment with tetramethylthiuram disulfide (thiram) (Arasan) did not control the Cercospora disease; all seeds were treated with it before planting but it did not help.

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[1]Department of Field and Vegetable Crops, Faculty of Agriculture, The Hebrew University, Rehovot, Israel.

[2]Ashri, Amram. 1961. The susceptibility of safflower varieties and species to several foliage diseases in Israel. Plant Disease Reptr. 45: 146-150.